The evolution of brain size and structure in primates

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Abstract

The pressures and constraints influencing the wide variation in primate brain size and composition are hotly contested. Comparative biologists have proposed many alternative hypotheses with no consensus yet emerging. This thesis uses phylogenetic comparative techniques and new data to explore the core issues in primate brain evolution; examining how behavioural ecology is associated with brain size and structure variation and what life history correlates reveal about possible developmental mechanisms producing this variation.

The thesis raises a number of important issues for the field. Firstly, evidence of selection at the level of individual structures independently of overall brain size further challenges the utility of whole brain size as a meaningful measure in comparative enquiry. Secondly, by analysing multiple datasets, I demonstrate that fluctuations in data quality are a major cause of inconsistency in results. Finally, the pursuit of explanatory frameworks based on single niche dimensions appears to yield unclear results; contributing to the lack of consensus in the literature. The concept of adaptive syndromes of correlates, while more difficult to operationalise, is likely more meaningful in terms of selection on function.

The findings demonstrate different patterns of covariation of structures across orders and varying correlates of individual structures within primates. This suggests that primate brain evolution has been characterised by the mosaic evolution of individual structures in response to ecological, social and developmental factors, and that selection on function is the primary cause of the observed phenotypic variation. Life history traits were also associated with structure size in a manner predicted from their developmental trajectories, suggesting that selection induces variation in brain composition by modifying the duration of specific life history phases to adjust the relative growth of individual structures.

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Declaration

Chapter 3 of this thesis "Re-evaluating the link between brain size and behavioural ecology in primates" has been published as a research paper and is the product of joint work between the thesis author Lauren Elizabeth Powell (LP), Professor Rob Barton (RB) at Durham University and Dr Karin Isler (KI) at the University of Zurich. RB and LP conceived of the project and wrote the manuscript; LP and KI collected the data; LP analysed the data. The rest of this thesis is the sole work of LP.

Statement of copyright

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1 Introduction

Relative to body size, primate brains are among the largest in Mammalia (Fleagle, 2013; Montgomery *et al.*, 2013) and exhibit a trend towards increased relative size over time across the primate phylogeny (Montgomery *et al.*, 2010). Despite this evidence of strong directional selection, absolute brain size in extant primates varies almost a thousand-fold across the order (Barton, 2012). The adaptive significance of this variation has been the subject of intense interest and is the focus of this thesis.

This thesis examines the large-scale patterns in primate brain evolution, exploring and expanding on fundamental ideas proposed to explain these patterns, bringing up to date methods and data to bear on key questions: why do primates have large brains? Is a big brain linked to some specific functions? How are the costs of a large brain offset? How do large brains get large?

1.1 Background

1.1.1 What is a big brain?

Primates have large brains and exhibit behaviours that are regarded as complex. The coincidence of these two traits has largely been taken as evidence for a causal, unidimensional relationship; so that a large brain is commonly assumed to be a proxy for complex cognition (Logan *et al.*, 2017). However, organ size tends to increase with body size. Larger animals therefore tend to have larger brains (Jerison, 1973). It was reasoned that a larger body requires more neural tissue to maintain somatic systems (Deaner, Nunn and van Schaik, 2000; Byrne and Corp, 2004; Chittka and Niven, 2009). Therefore, the large brains of large animals may not be indicative of any particularly increased cognitive capacity (Barton, 2012).

In response to this issue, Jerison (Jerison, 1973; Jerison and Barlow, 1985) proposed that the proportion of the brain that was in excess of that predicted by body size was surplus to the basic maintenance requirements of the body and so could be linked to "intelligence"[*]. He termed this the "encephalisation quotient" (EQ), with species whose brains were substantially

[*] This term is often avoided by those studying comparative cognition due to its anthropocentric and nebulous nature (Deacon, 1990). It is used here only in reference to historic ideas.

larger than that predicted by body size being more "encephalised" than those with more modest surpluses. This method of using the residual brain volume from an expected brain to body size relationship to examine neural differences across clades became a popular way of controlling for brain–body allometry.

However, the measure is problematic, as follows. To determine how encephalised a taxon is, a reference group must be used to provide an expected allometric exponent against which the observed values can be compared (Harvey and Krebs, 1990). These exponents have been shown to vary, both amongst taxa and according to the method of analysis used (Deacon, 1990; Harvey and Pagel, 1991; Barton, 2006c; Willemet, 2012). In the absence of a single mammalian exponent, the encephalisation quotient of a given taxon is variable depending on the reference group used. The EQ, and other indices based on such broad scaling relationships, are therefore of limited utility when comparing taxa from diverse taxonomic groups (Deacon, 1990; Martin, 1996). Nevertheless, Jerison's idea suggested that absolute brain size was potentially not useful in terms of explaining differences in cognition between taxa. Various measures of relative brain size (brain size relative to body size) have therefore largely become the measure of choice when seeking to examine meaningful brain size variation comparatively (Chittka and Niven, 2009).

Yet, there is still support for absolute brain size being a functionally meaningful measure. This measure has been reported to correlate with a number of (slightly ambiguous) behavioural measures such as self-control (MacLean *et al.*, 2012; Stevens, 2014), innovation and social learning (Reader and Laland, 2002), and "general cognitive ability" (Deaner *et al.*, 2007; Reader, Hager and Laland, 2011) (also referred to as "domain general intelligence" or simply "g"). The proposed functional reason for this is that absolute brain size represents neural complexity; larger brains have more neurons which requires increased modularity to maintain speed of connectivity (Marino, 2006). This increased complexity of organisation is proposed to give rise to increased cognitive capacity and more functional differentiation (Chittka and Niven, 2009; MacLean *et al.*, 2014). Absolute brain size also explains apparent differences between clades better than relative brain size (Deaner, Nunn and van Schaik, 2000; Striedter, 2006; Deaner *et al.*, 2007), but this has often been attributable to a lack of phylogenetic correction (Parker, 2015) (explained in "Approach" below). However, despite these possible advantages, it remains difficult to see how absolute brain size could be meaningful in terms of comparing cognition when compared across a number of taxa with different brain-body allometric relationships. This issue is particularly apparent when

considering taxa with very small absolute brain sizes but an impressive repertoire of cognitive abilities, such as honeybees (Chittka and Niven, 2009). Across large phylogenetic distances, cognitively meaningful differences in absolute brain size seem to break down.

1.1.2 What are big brains for?

Increasing brain size is accompanied by a variety of costs which must be compensated by benefits. Neural tissue is energy-hungry (Aiello and Wheeler, 1995). As a brain gets larger, its energetic needs increase. If the energetic needs of the rest of the body are held constant, there is an energy deficit to be paid. Aiello and Wheeler suggested that the cost of an increase in brain tissue must be traded-off against a decrease in other expensive tissues, in this case the gut (Aiello and Wheeler, 1995). A more recent elaboration of this kind of trade-off hypothesis is the Expensive Brain Hypothesis (Isler and van Schaik, 2009), which suggests that any of a broad range of energetically expensive traits may be traded-off in order to afford the cost of an increase in neural tissue, including reproduction and locomotion. Given these costs and trade-offs, it is reasoned that increases in brain size must confer some adaptive advantage on their owners. In order to discover the nature of these advantages, we can examine patterns of correlation between various brain measures and variables which represent potential selective pressures on cognitive function. Since we cannot directly measure cognitive function, behaviours are used as proxies (Logan *et al.*, 2017). This is common practice in comparative work which examines complex cognitive traits; for example, in their 2014 paper MacLean et al. used two behavioural tests based around response inhibition as proxies for the cognitive trait of "self-control" (2014).

The relationship between cognition and behaviour is widely debated in the psychological literature. The nature of their relationship is outside the scope of this thesis, but the use of both terms throughout this work warrants a brief justification. Recent work has emphasised the embodied nature of cognition, where cognition, sensorimotor responses (that might commonly be referred to as "behaviour") and the context in which they take place are integrated, and so ultimately cognition, behaviour and the context they occur in are indivisible (Barton, 2012; Keijzer and Keijzer, 2017).

Therefore where "cognition" and "behaviour" are referred to in this thesis, they should be understood as different aspects of the same process: one refers to a specific, discrete and observable action or collection of actions (behaviour), whereas the other is a more abstract term under which a number of overt actions may be defined (cognition). For example, in the

case of MacLean et al. (2014) the success of suppressing the impulse to reach for food (i.e. not reaching) is the behaviour, but the wider umbrella term that this action may represent is proposed to be "self-control" – a "cognitive" principle. The two are not necessarily distinct. The use of the two terms in this thesis should not be taken as support for their conceptual separation; rather they are used for ease of reference to these two levels.

1.1.2.1 Ecological Brains

Hypotheses seeking to explain the kinds of factors that can influence brain size and composition can be broadly broken down in to ecological, social, and developmental.

Ecological hypotheses (sometimes referred to as foraging hypotheses (Barton, 2006c; Mars *et al.*, 2014) suggest that a species' ecological niche may exert selective pressures on brain and behaviour (Harvey and Rambaut, 2000). The ecological niche concept was initially defined by Hutchison (1957) as a multidimensional space whose parameters are defined by ecological variables and delimit the possible habitat for a given species (Basille *et al.*, 2008). A species' niche is therefore a complex interaction of a number of ecological variables. Early comparative studies showed that brain size correlates with some of these ecological characteristics after accounting for variation associated with body size (Eisenberg and Wilson, 1978; Clutton-Brock and Harvey, 1980; Harvey, Clutton-Brock and Mace, 1980; Martin, 1984).

1.1.2.1.1 Diet

Many of the ecological hypotheses associated with brain size variation are concerned with food. Diet has been found to have an effect on brain size in a number of mammalian groups, including primates, carnivorans, rodents, lagomorphs and "insectivores" (Harvey, Clutton-Brock and Mace, 1980; Fish and Lockwood, 2003; Walker *et al.*, 2006; van Woerden, van Schaik and Isler, 2010; Swanson *et al.*, 2012; DeCasien *et al.*, 2017). Clutton-Brock and Harvey (1980) showed that folivorous primates tended to have smaller relative brain sizes than frugivores (and that home range size correlated positively with relative brain size – see below). A similar pattern has also been demonstrated in bats (Eisenberg and Wilson, 1978). Frugivorous chiropterans had larger relative brain sizes, with insectivorous species having the smallest brains relative to body size and the other dietary specialisations falling somewhere between the two (Martin, 1984). A pattern of folivorous species having comparatively small brains has also been observed in a number of small mammal taxa including Rodentia, Soricomorpha and Lagomorpha (Harvey, Clutton-Brock and Mace, 1980).

Diet is a large component of ecological hypotheses, but it is embedded in a wider ecological context that encompasses a range of other variables which have also been found to influence brain size. A number of hypotheses regarding the selection pressures and constraints responsible for this correlation between brain size and diet have been suggested. Finding food (spatial mapping/cognitive mapping), gaining access to it (extractive foraging), having enough of it to fuel the brain (the expensive brain) or coping with its scarcity or unpredictability in its distribution (cognitive buffering) are the main hypotheses which have been developed to explain this relationship.

1.1.2.1.2 Finding food

The 'spatial mapping hypothesis' interprets folivores' comparatively smaller brains as being due to the lower cognitive demands imposed by the predictable and even distribution of their food sources (Milton, 1988). For frugivorous species the increased navigation and spatial memory demands associated with unpredictable food distribution and consequent foraging strategies may require more neural tissue (Clutton-Brock and Harvey, 1980; Harvey, Clutton-Brock and Mace, 1980). The positive correlation between relative brain size and home range size (Clutton-Brock and Harvey, 1980; Dunbar and Shultz, 2007a) is consistent with this interpretation. The spatial mapping hypothesis therefore suggests that the relationship between brain size and diet may be indirect, with the direct effect due to the increased cognitive demands of navigating a large home range (Milton and May, 1976; Parker, 2015). The size of an animal's home range is largely influenced by its energetic demands; i.e. a consequence of the animal's body size, the type of food sources it exploits and the distribution of those sources (Mcnab, 1963). This three-way association between diet, home range size and brain size is suggested to support encephalisation as a response to the cognitive demands imposed by the environment rather than a constraint of the energetic value of the diet (Harvey and Krebs, 1990). It should be noted however that home range size and energetic demands do not scale in the same way across all primates due to confounding factors like terrestriality/arboreality, diet and home range overlap (Nunn and Barton, 2000).

1.1.2.1.3 Gaining access to hidden or protected food sources

Some species specialise in feeding on defended food sources which are difficult to extract, such as hard nuts and seeds and nest-dwelling insects. The extractive foraging hypothesis suggests that the complex sensorimotor cognitive processes necessary to overcome these obstacles and gain access to the consumable items could possibly be associated with brain size variation (Parker and Gibson, 1977; Parker, 2015). Extractive foraging, both with and

without tools, requires fine manipulation skills. Recent work by Heldstab et al. suggests an association between brain size and manipulative complexity which interacts with terrestriality (Heldstab *et al.*, 2016). They report that larger brained, terrestrial primate species tended to exhibit more complex manipulative skills, as measured by a nested ranking system of food processing behaviours. They suggest that the greater variety in available tools on the ground and the reduced need for highly specialised forelimb morphology associated with an arboreal way of life may be explanatory factors. Substrate use (in terms of arboreality versus terrestriality) has been found to have a relationship with relative brain size, with terrestrial species tending to have larger brains relative to body size (Sawaguchi, 1990). Terrestriality may therefore be indirectly associated with increased brain size through manipulation complexity.

1.1.2.1.4 Predicting food availability

In addition to the problem of locating and gaining access to food, some species also face the problem of unpredictable distribution of food. This is commonly due to seasonal fluctuations in abundance. Two hypotheses interpret the effects of unpredictability in food distribution in different ways. The Cognitive Buffer hypothesis was postulated to explain correlations between brain size and lifespan. It suggests that unpredictability in food availability positively selects for larger brains as this allows for increased behavioural flexibility (a "cognitive buffer") which enables the animal to effectively manage fluctuations in food availability by utilising defended or cryptic food sources or switching to different "microhabitats" (van Woerden, van Schaik and Isler, 2010; van Woerden *et al.*, 2012).

However, The Expensive Brain Hypothesis (Isler and van Schaik, 2009) takes a different perspective; dietary correlates are interpreted as a reflection of constraints on brain size (Aiello and Wheeler, 1995; Fish and Lockwood, 2003). This model grew from Martin's Maternal Energy Hypothesis (Martin, 1996) which suggested a link between infant brain size and the energetic contribution from the mother (measured by basal metabolic rate and gestation length) across mammals (see also Barton & Capellini (2011)). The Maternal Energy and Expensive Brain Hypotheses predict that food scarcity will limit brain size (or force the animal to make compensatory changes in energy usage, for example in limiting energy expenditure on reproduction). Under this hypothesis, the larger brains of frugivores are explained by the higher energetic content per unit of fruit relative to that of leaves providing more energy for brain growth. Results consistent with these models have been obtained in Carnivora, where carnivorous species tend to have larger brains than their insectivorous and

omnivorous counterparts whose food sources may not be as energetically rich (Swanson *et al.*, 2012), and across mammals (Barton and Capellini, 2011; van Woerden *et al.*, 2012; van Woerden, van Schaik and Isler, 2014).

Models which emphasise energetic costs and cognitive buffering models are not necessarily mutually exclusive; it is possible that if unpredictable food distribution drives brain size up, it also acts as a limiting factor given the amount of energy that can be extracted from the diet is finite. To take an example: Van Woerden et al. (2012) argued that the apparent effect of seasonality on catarrhine brain size affected brain size both as a constraint and selective pressure. Species in more seasonal habitats had smaller brains, which was interpreted as reflecting the energetic costs of seasonal fluctuations in food availability limiting brain size. However, larger brained species displayed more behavioural flexibility in overcoming the challenges associated with seasonality, suggesting that the unpredictable habitat may select for larger brains. This is presented as support for 'The Cognitive Buffer Hypothesis'.

The issue of distinguishing constraints from selection pressures is important to note. When examining correlates across species, we cannot reliably infer the direction of causation. As made clear by the example above, we cannot determine whether a correlation between change in a certain brain area and a dietary variable is due to the influence of selection acting upon a function mediated by that area that is adaptive for that dietary niche, or whether the energetic value of that dietary strategy is a constraining factor on neural change. This has been the basis of some disagreement over the relative importance of predictors of brain size in the literature (Dunbar and Shultz, 2017). The wider issue of causation and its discussion in the literature is dealt with in Chapter 6, but it is important for now to note both the distinction between pressures and constraints and our inability to differentiate them with only correlational analyses.

1.1.2.1.5 Activity period and the visual brain

The timing of species' waking hours also influences brain size and composition. Primates are a diverse order in terms of activity patterns, including diurnal, nocturnal and cathemeral lineages. Activity period has been shown to relate to brain structure evolution, with olfactory structure enlargement in nocturnal lineages and visual system enlargement in diurnal taxa (Barton, Purvis and Harvey, 1995). Diurnal primates have larger neocortices than do nocturnal primates (Barton, 1996). This pattern is proposed to be linked to the large proportion of cortex associated with vision in primates. This led Barton (2007) to formulate a

hypothesis which explains variation in primate brain size based on visual specialisation. He suggests that a large amount of variation in cortex size, and so brain size, is attributable to species' relative investment in visual adaptations. Primate cortex volume is associated with the volume of the parvocellular system of the lateral geniculate nucleus, which facilitates colour discrimination and high acuity stereoscopic vision (Barton, 1998, 2004). This, coupled with the correlation of frugivory and activity period with the parvocellular system (Barton, 1998), led Barton to suggest that these visual adaptations served to enable high acuity photic vision and thence the capacities to distinguish, identify and process ripe fruit (Barton, Purvis and Harvey, 1995; Barton, 1998, 2004).

1.1.2.2 The Social Brain

The cognitive demands of managing different social relationships amongst conspecifics has been suggested as an important selective pressure on primate intelligence (Jolly, 1966; Humphrey, 1976; Whiten and Byrne, 1988). Clutton-Brock and Harvey (1977) had acknowledged a potential social influence on primate brain size, including mating system as a predictor variable. They reported that monogamous primates have smaller brains than polygynous species (Clutton-Brock and Harvey, 1980); a finding later replicated by Sawaguchi (1990). These comparative analyses find a corollary in Brothers' 1990 hypothesis that anthropoid primate brains are specialised for social cognition (Brothers, 1990).

Dunbar (1992) built on these insights, using comparative analysis to argue that it was sociality, rather than ecology, that appeared to have been the primary driver of the evolution of large brains. He argued that the ecological models had not been robustly compared with alternative hypotheses and pointed out that diet and home range size both correlate with group size, potentially confounding previous results. He saw a weakness in ecological explanations of brain size evolution, in failing to account for large differences in brain size between organisms occupying similar niches. Comparing social and ecological hypotheses, he examined the relationship between relative neocortex size and ranging, frugivory and group size. He chose to focus on the neocortex rather than the whole brain as; a) it makes up a large proportion (50 to 80%) of the primate brain, and b) it has changed independently of more conserved structures like the medulla (Dunbar, 1998). This choice also appears to have been influenced by the commonly held assumption that the neocortex is "..the seat of those cognitive processes that we associate with reasoning and consciousness.." (p.181). His study found a relationship between sociality and neocortex size, and no such relationship for

ecological variables associated with ranging and diet (Dunbar, 1992). This neocortical dominance has reigned in comparative brain work ever since.

Following these analyses, Dunbar and colleagues formally set out the Social Brain Hypothesis (Barton and Dunbar, 1997; Dunbar, 1998), which had grown from Brothers' paper (Brothers, 1990) and has dominated discussion of the behavioural ecology of brain size since. This hypothesis proposed that increased social complexity imposes larger cognitive demands through having to manage and maintain multiple social bonds, therefore exerting a selective pressure on brain size. The hypothesis has been widely supported (Barton, 1996; Kudo and Dunbar, 2001; Reader and Laland, 2002; Byrne and Corp, 2004; Walker *et al.*, 2006; Sallet *et al.*, 2011; Powell *et al.*, 2012; Arsznov and Sakai, 2013), not least by the observation that primates which live in larger groups tend to have larger brains (Barton and Dunbar, 1997). The relationship has also been found in other mammalian taxa such as Ungulates (Shultz and Dunbar, 2006) and Carnivora (Pérez-Barbería, Shultz and Dunbar, 2007; Swanson *et al.*, 2012; Holekamp *et al.*, 2015). Social complexity has most frequently been measured by group size (Barton, 1996; Dunbar, 1998; Kudo and Dunbar, 2001; Lehmann, Korstjens and Dunbar, 2007; Dunbar and Shultz, 2007a), but has also been represented by deception (Byrne and Corp, 2004), social system (Barton, 2006b), mating system (Barton, 2006b), and play prevalence and complexity (Pellis and Iwaniuk, 2002).

However, recently there has been some disagreement over the broad explanatory value of this apparent relationship between sociality and brain size. It is not clear how grade-shifts (van Schaik *et al.*, 2012) in brain size, not accompanied by obvious differences in sociality, can be readily accommodated by the hypothesis. As Byrne (2006) points out, monkeys often live in much larger groups than apes, but their brain size is systematically smaller in absolute size. In the same vein, relatively small brained but social animals like spotted hyaenas (*Crocuta crocuta*) and relatively large brained but solitary animals like orangutans (genus *Pongo*), aye-ayes (*Daubentonia madagascariensis*) (Holekamp, 2007), and mustelids (Swanson *et al.*, 2012) represent significant challenges to the idea that sociality is the primary driver of brain size evolution.

1.1.3 How brains evolve

The study of brain evolution is concerned with how brains vary between taxa. But by what mechanism does this variation occur? Some propose that selective pressures cannot act directly on brain structures and systems because they are constrained by developmental

factors which govern the composition of the brain. Therefore, selective pressures can only change individual structures in concert with others. This "concerted evolution" model was advanced by Finlay and Darlington (1995), who emphasised the universality of this pattern across mammalian brain evolution.

However, evidence of specific differences in brain composition between clades has undermined this point of view. Such "grade shifts" show that the size of structures relative to one another can vary substantially across taxa; an outcome which is arguably not possible in the original concerted evolution model, which postulates only approximately two-fold variation in component size independent of the global constraint (Finlay and Darlington, 1995, p. 1580). Furthermore, observations of covariation between the size of individual brain components and external factors, independent of the size of the rest of the brain have demonstrated that selection can influence parts of the brain separately from the whole. This process by which selection pressures may exert themselves differently on different structures or groups of functionally or anatomically connected structures leading to their independent evolution separate from evolutionary changes in gross brain size is known as 'mosaic evolution' (Barton and Harvey, 2000). This model holds that, rather than being constrained by overall brain size, individual structures vary according to selection. A classic example of this model is the relative enlargement of visual structures and diminution of olfactory structures in diurnal primates (Barton, Purvis and Harvey, 1995). The converse is observed for nocturnal species. This example shows how the pressures associated with a species' niche interact directly with the neural machinery mediating function. Experimental studies have also demonstrated that selection can act directly on brain size and structure (Kolb *et al.*, 2013; Kotrschal *et al.*, 2013). If primate brains have changed more by a pattern of mosaic evolution rather than purely global brain size changes, we should see more pronounced effects of selective pressures on individual brain structure than overall brain size (Montgomery, Mundy and Barton, 2016). This debate will be examined in more detail in Chapter 2.

1.1.4 Brain composition and neocortical domination

An intrinsic problem with examining correlates of brain size is that the brain is composed of many structures of heterogeneous morphology and function (Harvey and Krebs, 1990). While cognitive functions are often distributed across multiple neural structures and one structure may mediate several different cognitive processes (Buckner and Krienen, 2013; Ribeiro *et al.*, 2013; Montgomery, Mundy and Barton, 2016), there is some degree of modularity in the brain with certain structures or groups of structures displaying some functional specificity

(Kanwisher, 2010; Mars *et al.*, 2013). This presents a challenge to detecting changes in whole brain size associated with variables of interest as these functional complexes of structures change size at different rates in response to different selective pressures (Barton and Harvey, 2000). In addition to this, growth in one structure (or set of functionally linked structures) may be masked by reduction in another, resulting in no detectable change in overall brain size (Barton, Purvis and Harvey, 1995; Barton, 1999; Healy and Rowe, 2007). Therefore, it is arguably more valid to explore behavioural correlates of individual structures than of whole brain size.

Since the neocortex accounts for a large proportion of the primate brain and is relatively large in comparison to other taxa, it has been a central focus of the study of variation in brain structures. The confluence of large neocortices and complex cognition in primates led many to assume a causal relationship and hold the neocortex as the seat of so-called "higher" cognition and so the pinnacle of brain evolution (Barton, 2012). Dunbar (1992) asserted that neocortical variation is the source of most interspecific variation, and that the neocortex is "the 'thinking' part of the brain" (Dunbar, 1992, p. 473). He therefore treated neocortical volume as an "anatomical index for cognitive capacity" (ibid.). It has been heavily linked to his Social Brain hypothesis (Dunbar, 1992; Aiello and Dunbar, 1993; Barton, 1996; Dunbar, 1998; Kudo and Dunbar, 2001; Reader and Laland, 2002; Byrne and Corp, 2004; Shultz and Dunbar, 2006), but it has also been the focus of many studies of ecological, technical and life history correlates of brain variation (Eisenberg and Wilson, 1978; Barton, 1996, 1998, 2007; Deaner, Nunn and van Schaik, 2000; Walker *et al.*, 2006; Padberg *et al.*, 2007; Heldstab *et al.*, 2016). The neocortex has therefore long reigned supreme as the structure of primary importance in explaining mammalian brain evolution.

The dominance of the neocortex in the literature is being challenged, however. Changes in the neocortex lead to much greater overall change in brain size than other structures as it makes up a large proportion of the brain and scales hyperallometrically with it (Logan *et al.*, 2017). However, cortical expansion is associated with a disproportionate increase in the proportion of white matter to grey matter, suggesting that the size increase is more due to conservation of neural connectivity rather than an adaptive change (Barton, 2012). Neocortical expansion therefore does not necessarily represent a simple linear increase in cognitive capacity as implied by some cortico-centric studies.

The focus on the importance of this single structure is most notably challenged by its intimate connection with the cerebellum. The cerebellum, formerly considered to be "simply" a motor structure, is being increasingly linked with cognitive functions previously attributed to the cortex (Leiner, Leiner and Dow, 1993; Cantalupo and Hopkins, 2010; Smaers, Steele and Zilles, 2011). The mammalian neocortex and cerebellum have undergone correlated volumetric evolution (Whiting and Barton, 2003; Barton, 2012) and form a functional system, with reciprocal loops between the two (Ramnani, 2006; Kipping *et al.*, 2013; Koziol *et al.*, 2013). The coordinated growth of these two structures heavily influences brain size and composition variation (Barton, 2006c, 2012; Smaers, Steele and Zilles, 2011). The examination of neocortical variation is therefore arguably incomplete without consideration of concomitant cerebellar variation, and both structures are of equal importance in the discussion of overall brain evolution (Barrett, Henzi and Lusseau, 2012; Barton, 2012).

1.2 Aims

This thesis aims to re-examine these major issues in brain evolution, which have been dominated by whole brain and neocortex studies, in the light of newer ideas about mosaic brain evolution using updated techniques and better quality, more recent data. Despite a long and prolific history, the literature on the forces which have shaped primate brain evolution remains remarkably equivocal, with even the most fundamental issues still somewhat polarised. This is exemplified by the debates surrounding ecological versus social and concerted versus mosaic explanations of brain evolution. This polarisation has often obfuscated debate.

This work will aim to update and extend the current literature in the following ways:

1. Acknowledge the heterogeneous nature of the brain, examining the effects of pressures/constraints shaping brains by looking at more functionally specific sub-structures and how their relationships relate to major whole-brain and neocortex focused hypotheses
    a. Examine the behavioural-ecological correlates of brain size and composition variation
    b. Test hypotheses based on life history correlates of brain size using specific structures and their developmental trajectories

c. Test predictions of variation in brain composition based on developmental constraints and selection on function

2. Examine possible interdependence between traits related to brain variation which have previously been treated as independent
3. Test for potential variability in results and identify causes of disagreement in the literature surrounding ecological and social hypotheses explaining brain size and structure variation
4. Revisit long-standing ideas and apply appropriate phylogenetic comparative techniques and size correction.

1.3 Approach

1.3.1 Phylogenetic comparative methods

A core assumption of ordinary statistical techniques used to explore the covariation of traits such as regression, ANOVA and principal components analysis, is that of the independence of data points. Put simply, the data are assumed to have no underlying structure. In comparative analyses the data points represent species values. Since all species are phylogenetically related to varying degrees, these data cannot be said to be truly independent (Freckleton, Harvey and Pagel, 2002). In comparative brain evolution, we are often interested in detecting factors which may have influenced a given trait. A shared trait may be due to the common influence of an external factor, such as the behavioural, ecological, social and developmental variables explored in this thesis, or it could be due to the taxa sharing an inherited trait from a common ancestor (Nunn and Barton, 2001). It is therefore necessary to control for the degree of phylogenetic relatedness between species.

Phylogenetic comparative methods enable the user to incorporate a phylogeny in to a model, thereby controlling for phylogenetic effects. They can also be used to estimate ancestral conditions at specific nodes in a phylogenetic tree, thereby enabling us to test predictions about extinct taxa. However, in this thesis, these methods are primarily used to detect correlated evolution between traits. This thesis predominantly uses phylogenetic least squares regression (PGLS) to achieve this. In PGLS, a variance-covariance matrix is derived from a phylogenetic tree which describes not only the topology of relatedness between species but also branch lengths which give a measure of phylogenetic distance in terms of time since a last common ancestor, or in terms of genetic distance between taxa (Nunn, 2011). This

information can then be incorporated in to the error term of the regression model, thereby controlling for phylogenetic nonindependence. The degree to which covariance in species' traits reflects their phylogenetic relatedness is estimated by the parameter λ. Principal components analysis is also used in this thesis, and phylogeny is corrected for in the same way in preliminary transformations (Revell, 2009). These methods are dealt with in more detail in the chapters in which they are used.

1.3.2 Size correction

As discussed above, body mass and brain mass are (allometrically) correlated (Jerison, 1973; Harvey, Clutton-Brock and Mace, 1980; Barton, 2006c). Most of the variation in brain size is attributable to body size variation (Jerison, 1973). Since many studies seek to test relationships between brain size and a hypothesised predictor variable, this potentially confounding variable is controlled for to avoid spurious relationships between predictors and body mass rather than the dependent variable (Barrickman *et al.*, 2008). This has often been achieved by using a measure of brain size that is relative to body size, rather than absolute size. A number of different methods for size correction have been employed in comparative studies of brain size and the literature is equivocal as to which is the most appropriate (Deaner, Nunn and van Schaik, 2000).

Some brain researchers have used ratios or proportions, such as neocortical ratio (Dunbar, 1992) or "executive brain" ratio (Reader and Laland, 2002). These measures are prone to bias, as they cannot distinguish allometric change from selection driven change which is independent of size (Barton, 2002). As previously mentioned, a common method is to observe how a species' data point might diverge from the predicted allometric relationship between body and brain size and use that value as data (Freckleton, 2002). One variant of this method is the analysis of residuals. In order to address the problem of varying allometric exponents between taxa, the exponent is estimated empirically for the species in the dataset using a least squares regression of log body size on log brain size (Rilling and Insel, 1999; Barrickman *et al.*, 2008; González-Lagos, Sol and Reader, 2010; Nunn, 2011). Once the exponent is defined and the residuals are obtained, the residuals are then used as data to allow subsequent analysis of relationships between relative brain size and traits of interest. However, a number of papers reviewing this use of residuals have criticised the practice (Garcia-Berthou, 2001; Freckleton, 2002). They point out that in fields such as comparative biology, where predictor variables are often collinear, using residuals can lead to systematic bias in parameter estimates, misallocation of degrees of freedom and instability of models

associated with the order of the entry of predictors. They suggest a more reliable way of controlling for body size is to include it as a predictor variable in a multiple regression. Including body size as a predictor in the model does not require any a priori estimation of any overall scaling exponent (Deaner *et al.*, 2007) which as previously mentioned is not stable across taxa. It also includes all of the predictors simultaneously, removing a potential source of instability. This thesis will utilise this method.

Body size itself is a problematic measure as it does not scale linearly with brain size (Logan *et al.*, 2017) and varies both between and within individuals due to factors such as life history, nutrition, and sex (Harvey and Krebs, 1990; Barrickman *et al.*, 2008). It has been suggested that poor estimates of body size can produce false positive or negative results by biasing predictors in the same direction as the measurement error (Barrickman *et al.*, 2008). This has led some to seek structures whose size variation is relatively evolutionarily conserved such as the brain stem (Dunbar, 1992; Reader and Laland, 2002) and spinal cord (Willemet, 2013) for size correction. However, comparative data on these structures are still relatively sparse and do not afford the taxonomic breadth required for large scale comparative analyses. Therefore, whilst acknowledging its possible shortcomings, body size is used for size correction in this thesis.

1.3.3 Volumetric measures

Volumetric measures are the foundation of comparative brain studies. They have been extensively, and until relatively recently, exclusively used to investigate correlated change between brains and behaviour. However, they are problematic as there is often no explicit, definitive explanation of what an increase or decrease in the volume of a structure means functionally (Healy and Rowe, 2007). The (most often tacit) assumption from which brain size studies proceed is that the adaptive advantage of a larger brain is some form of increased cognitive potential; larger brains are assumed to have greater computational and cognitive capacity (Healy and Rowe, 2007; Weisbecker *et al.*, 2015). Brain size is therefore treated as "an anatomical proxy for cognitive ability" (MacLean *et al.*, 2012).

Further, examining aspects of a species' lifestyle which correlate with brain or brain structure size is thought to illuminate the selection pressures responsible for their variation. Put differently, the differences between species in terms of their brain composition is deemed to reflect adaptation to their respective niches. Consequently, size increase in a brain area is deemed to be the manifestation of increased computational power in that region, which is

assumed to be necessary to cognitively manage the challenge imposed by the correlated aspect of the niche. Jerison termed this 'the principle of proper mass' (Jerison, 1973). However, the relationship between the volume of a structure and its neuronal density is variable across the brain and across species (Herculano-Houzel, 2012; Herculano-Houzel, Manger and Kaas, 2014). Thus, an increase in the size of two different structures (or the same structure in two different species) does not necessarily indicate an identical increase in computational power. It is therefore likely that much more informative variation can be uncovered with more direct measures of computation like neuron density or number (Roth and Dicke, 2005). This subject is dealt with in more detail in Chapter 6.

Despite these caveats, the examples given in "Background" above demonstrate that volumetric measures have been shown to correlate with behaviour in line with predicted relationships. This suggests that volumetric measures are still useful for identifying large scale patterns. In addition, the scarcity of comparative data for neuroanatomical measures at finer scales such as neuron number precludes their inclusion in large scale comparative analyses like those undertaken in this thesis. Since this thesis also endeavours to re-visit previous results and hypotheses, using volumetric measures facilitates comparison with these sources.

1.4 Structure of thesis

Chapter 2 begins the thesis with an examination of the multivariate structure of mammalian brain size. Phylogenetic principal components analysis and least squares regression are employed to examine commonalities and divergences in brain composition between three mammalian orders. It identifies primates as a taxon with a pattern of brain composition that is distinct from the other taxa analysed and justifies the focus of rest of the thesis on this order.

Chapter 3 explores the two main umbrella hypotheses which have dominated the discussion of primate brain evolution; the ecological brain and the social brain. It scrutinises the reasons for the ongoing lack of consensus in this area, comparing results from different datasets and modifying samples to simulate different sources of variation. This chapter has been published as a research paper (Powell, Isler and Barton, 2017).

Chapter 4 builds on the previous chapter's findings at a finer scale by evaluating the behavioural-ecological correlates of variation in brain structure volume. New brain volume

data which updates and augments an existing well-known dataset is incorporated and differences in results between datasets is again examined.

Chapter 5 concludes the empirical component of the thesis by investigating how the costs and benefits of variation in brain size and composition are balanced in terms of life history. The life history correlates of brain composition are analysed in the context of the developmental scheduling of individual structures and major hypotheses relating to energetic constraints and cognitive buffering.

A summary and conclusions chapter ends the thesis, bringing together the major threads, examining the limitations of the findings and providing reflections for future research.

2 The position of primates within the multivariate structure of the mammalian brain

2.1 Introduction

The volume of brain structures and their relative size in proportion to total brain volume varies widely between mammalian species. What causes brain structure sizes to vary in this way? Previous work addressing this question has broadly addressed two models; concerted and mosaic evolution. The concerted evolution model proposes a universal mammalian pattern of brain composition in which brain structures vary together according to allometric scaling defined by developmental constraints, characterising the brain as a "coordinated processing device" (Kaskan *et al.*, 2005; Yopak *et al.*, 2010) . In contrast, the mosaic evolution model suggests that individual structures and/or systems can vary in size independently of each other and the rest of the brain according to selection on specific functions.

2.1.1 Concerted brain evolution

Concerted brain evolution emphasises the conserved nature of developmental processes on the brain. This ontogeny focused theory was popularised by Finlay and Darlington (1995). Their paper argued for a conserved pattern of structure growth in mammals, constrained by overall brain development mechanisms. This hypothesis grew from the observation that the size of a given gross brain structure is strongly predicted by overall brain size (Finlay and Darlington, 1995; Yopak *et al.*, 2010). The hypothesis suggests that structure size change is primarily generated by changes in allometric scaling (Striedter, 2006), and not due to direct selection on structure size or individual functional systems (Kaskan *et al.*, 2005; Barton, 2007). Therefore, evolutionary changes in structure size tend not to occur independently of the size of the rest of the brain. Finlay and Darlington argued that structures which increase in the proportion of brain that they comprise as overall brain size gets larger do so because the peak of their neurogenesis is later (than that of structures which are proportionally smaller), allowing more time for progenitor cell pools to generate. This larger "founder" pool generates more neurons and so causes the structure to grow disproportionately large (Charvet and Finlay, 2012). In this hypothesis, selection on cognitive function causing the size of the structure(s) to increase must be mediated by increasing the size of the whole brain.

2.1.2 Mosaic brain evolution

The size of a mammal's brain can be predicted with reasonable accuracy from the size of its body (Barton and Harvey, 2000; Roth and Dicke, 2005; Smaers and Soligo, 2013), such that larger animals tend to have larger brains than their smaller relatives (Buckner and Krienen, 2013). However, this scaling does not explain all of brain size variation (Barton and Harvey, 2000; Smaers and Soligo, 2013), as individual structures within the brain also vary in size. While individual structures have been found to scale with total brain size, they can also vary independently of overall brain size and of each other. It is suggested that this independent variation represents adaptive specialisation (Barton, 2007), in which changes in the size of structures are naturally selected based on their functional capacities (Harvey and Krebs, 1990; Healy and Harvey, 1990; Barton and Harvey, 2000). This has been observed in comparative studies of primates (Barton and Harvey, 2000; Smaers and Soligo, 2013), wasps (O'Donnell *et al.*, 2018), lizards (Hoops *et al.*, 2017) and experimentally tested in mice (Kolb *et al.*, 2013). Support has also come from genetic studies, demonstrating that structures' developmental scheduling is likely scheduled by structure-specific genes rather than genes coordinating global brain growth (Harrison and Montgomery, 2017; Li *et al.*, 2017).

Mosaic brain evolution also emphasises the role of selection in the coevolution of functionally related structures (Montgomery, Mundy and Barton, 2016). Function is often mediated by more than one structure and distributed across a number of areas (Buckner and Krienen, 2013). If selection on function drives volumetric changes in structures, then functionally linked systems of structures should change in tandem. This has indeed been observed: some structures show correlated volumetric change independent of changes in the size of the whole brain and other composite brain structures (de Winter and Oxnard, 2001; Barton, 2002; Whiting and Barton, 2003; Barton, 2012; Barton and Harvey, 2000). Mosaic brain evolution can therefore act at two levels: the level of individual anatomical structures and the level of the functionally linked systems of structures (Montgomery, Mundy and Barton, 2016).

One such functionally linked system is that of the corticocerebellar complex (Whiting and Barton, 2003). Discussion of brain evolution has been historically dominated by the neocortex, likely due to the fact that it appears to contribute most to the "encephalisation" (Jerison and Barlow, 1985) (i.e. brain volume in excess of that predicted by an allometric brain-to-body size exponent) of highly encephalised species, although this is likely due to the fact that change in larger structures result in larger changes in absolute brain size (Willemet, 2013). However, the literature is increasingly finding that a) the neocortex is neither functionally nor anatomically independent of subcortical areas, and b)

variation in subcortical structures has been just as important a force in brain evolution as variation in the neocortex. A structure that appears to have a particularly close relationship with the neocortex is the cerebellum. These structures have undergone correlated size evolution in the primates (Whiting and Barton, 2003) and in mammals more widely (Barton, 2012).

2.1.3 The divergence of concerted and mosaic perspectives

A major point of difference between concerted and mosaic brain evolution is arguably the role of function (Barton, 2006a). The concerted evolution hypothesis holds that selection on function does not influence structure sizes independently of the whole (Striedter, 2006). Function is therefore somewhat decoupled from a structure's (eventual) size (Charvet and Finlay, 2012). In the case of the cortex at least, some proponents even suggest that function is possibly only linked to structure after developmental processes have already determined structure composition (Kaskan *et al.*, 2005). Conversely, the mosaic brain evolution hypothesis holds function to be central to the shaping of the brain and its composite structures, with selection on function directly modifying the structures and systems which mediate it (Barton and Harvey, 2000; Montgomery, Mundy and Barton, 2016).

2.1.3.1 Explaining taxonomic differences

So-called "grade shifts" are relationships which differ in magnitude but not nature across clades. When plotted, they have the same slopes but differ in their intercepts according to taxonomic differences (Nunn and Barton, 2001). The larger volume of the cerebellum relative to the neocortex in apes than in non-apes (Barton and Venditti, 2014) is an example of such a grade shift. These shifts have been used as supporting evidence for the mosaic evolution perspective as they demonstrate that animals of a comparable body size, yet inhabiting different niches display different patterns of brain composition. The importance of grade shifts in brain composition was played down in the original 1995 incarnation of the concerted evolution model (Finlay and Darlington, 1995), which emphasised a possible role of intra-specific variation and measurement error in apparent large taxonomic differences (p. 1580). Since then, some elaborations of the hypothesis have incorporated grade shifts (referred to as "taxonomic differences"), but explain these with reference to differing rates of neurogenesis across taxa (Charvet, Striedter and Finlay, 2011; Charvet and Finlay, 2012) rather than functional selection. Thus, mosaic changes in brain composition which differ between clades are explained by selection acting to extend or curtail the duration of neurogenesis. Instead of interpreting developmental processes as consequences of selection on function, they are interpreted as causes of or constraints on structure variation. However, proponents still largely hold these clade-level changes in neurogenesis to be relatively rare and inconsequential in comparison to wider developmentally

constrained regularities (Striedter, 2006), and in some instances still appear to be sceptical of the existence of grade shifts (Kaskan *et al.*, 2005).

2.1.3.2 Correlated evolution of structures

The position of the concerted evolution hypothesis on the correlated volumetric evolution of structures is slightly difficult to characterise. On one hand, proponents suggest that structures vary only according to variation in developmental processes and so correlation between the evolutionary growth of structures is a result of allometric scaling (Finlay and Darlington, 1995; Kaskan *et al.*, 2005). On the other, they suggest that selection on function may have a role in modifying developmental processes, and so in the ultimate size of structures. The position of the mosaic hypothesis is much clearer; selection for function acts on functionally linked structures in concert.

2.1.4 Principal components analysis in the exploration of brain evolution

Principal components analysis (PCA) PCA is a dimension reduction method which finds a number of linear components present in a group of variables (Field, Miles and Field, 2012). These components are orthogonal to one another, meaning they are uncorrelated. The first component encompasses the largest amount of variance in the data. The second represents the largest amount of the remaining variance. The contribution of the original variables to these new components is then revealed through loadings, allowing interpretation of the meaning of the component. These properties allow large, multivariate datasets to be summarised by a few components which encompass the majority of the variance. PCA has been widely used to explore comparative brain structure and results have been used to support both concerted and mosaic perspectives (Finlay and Darlington, 1995; de Winter and Oxnard, 2001; Yopak *et al.*, 2010; Smaers and Soligo, 2013).

The PCA-based investigation of Finlay and Darlington uncovered evidence supportive of the concerted evolution model in mammals. This approach has since also been used to support extending the suggested universal conserved pattern from just the mammals to all of Gnathostomata (jawed vertebrates) (Yopak *et al.*, 2010). In contrast, Oxnard and de Winter (de Winter and Oxnard, 2001) used the same method but found different patterns of variation in different clades; species separated in the subspace according to taxonomy and ecological factors (e.g. diet, locomotion etc.). Their analysis showed the three investigated orders (primates, bats and insectivores) were well differentiated, stretching along almost orthogonal axes from each other (p. 710). However, like Finlay and Darlington, this study did not properly account for species' phylogenetic relatedness.

Smaers and Soligo (2013) also used a PCA approach to examine primates in particular. They reported results which supported a mosaic evolution interpretation. Components reflected linked

groups of structures, notably including the cortex and cerebellum. However, they used residuals from a phylogenetically controlled regression in order to control for allometric effects of body size on brain size and brain size on brain structure size. This approach can lead to systematic biases in parameter estimates, particularly in cases such as this where the controlled variables and the dependent variables are collinear and has been cautioned against (Freckleton, 2002, 2009; Nunn, 2011).

2.1.5 The present study

This chapter is an exploratory study, re-examining the evolutionary architecture of the mammalian brain with updated methodology. It explores similarities and differences in the dimensionality of brain structure across 3 eutherian mammal orders[†] using appropriate phylogenetic analysis and size correction methods. The data are explored using a phylogenetic principal component analysis (pPCA). This method should reveal whether species have radiated independently in terms of their brain composition, or whether they are constrained by size-related developmental parameters. If the former, the taxa should form independent groupings in the subspace. If the latter, they should all lay along one axis of variation which reflects size. If differences between orders are revealed, component loadings should allow the characterisation of the dimensions along which the taxa differ.

The pPCA is conducted in two conditions; one controlling for size by including body size (hereafter referred to as "relative condition") as a covariate and one with no size control (hereafter referred to as "absolute condition"). The relative condition should reveal how the dimensions of brain composition vary independently of body size. The absolute condition, having no size correction, should reveal change which is not independent of overall changes in size. The pPCA which includes body size is predicted to reveal a first component on to which all structures load heavily, representing an overall size dimension. Subsequent components should then reveal groups of structures which covary in their size evolution independently of body size. The difference in conditions should not affect results if brain composition evolves according to the principles of concerted evolution, since the inclusion of body size should make no difference as the model suggests that there is no meaningful variation left after size is accounted for. According to the concerted evolution model, in both conditions, the first component should represent size and there should be no other significant components. If brain evolution is mosaic, it is expected that the first component in the body size-controlled condition will again be size, but subsequent components should reveal loadings from structures which have evolved in a mosaic fashion, independently of

[†] The dataset includes the now defunct order Insectivora. This order has been shown to be polyphyletic but is still a useful grouping for discussion in this instance.

overall size. In the absolute condition, the components subsequent to the first should reveal loadings from structures which have coevolved but do not vary outside of allometric scaling.

Since the neocortex has undoubtedly been the most researched gross structure in brain evolution, and in the light of recent advances in our understanding of its relationship with the cerebellum, these two structures are analysed further using phylogenetic least squares regression (PGLS) to explore their patterns of correlated evolution with structures and how this might differ across clades. If certain structures have undergone correlated size evolution, a PGLS controlling for body size should recover a significant partial regression coefficient between them, while no such association should be present with other areas.

2.2 Methods

2.2.1 Data sources

Volumetric data was sourced from a widely used dataset (Stephan, Frahm and Baron, 1981) which describes brain structures for primates, bats and "insectivores"[‡]. While this dataset is not recent and has possible issues with the representation of some clades (Powell, Isler and Barton, 2017), it still offers the largest comparative mammalian sample of volumetric brain structure data collected by one group with a uniform methodology. The 3 orders were divided in to nine subtaxa. The primates were divided in to Hominoidea, Cercopithecoidea, Tarsiidae and Strepsirrhini, the bats in to Yinpterochiroptera and Yangochiroptera, and the insectivores in to Scandentia, Eulipotyphla and Afrotheria. As this exploratory analysis endeavoured to examine large-scale patterns in brain composition, the variables chosen where those for which data were available for the largest number of species. Three species were excluded due to missing records; both rodent species *Nannospalax ehrenbergi* and *Rattus norvegicus*, and the yinpterochiropteran (formerly megachiropteran) *Macroglossus minimus*. The resulting dataset included 9 neurovolumetric variables (mm^3) and body size (g) for a sample of 301 mammalian species. The neurovolumetric variables were whole brain, main olfactory bulb (MOB), medulla, hippocampus, amygdala, cerebellum, neocortex, striatum and septum. The phylogeny used in all analyses was the Bininda-Emonds mammalian supertree (Bininda-Emonds et al., 2007). Taxonomic mismatches were resolved using synonym searches on the IUCN Red List of threatened species website (International Union for Conservation of Nature

[‡] The dataset includes the now defunct order "Insectivora". This order has been shown to be polyphyletic but is retained in this analysis for the purposes of comparison with previous studies which have used it.

and Natural Resources., 2017) and the 10k Trees taxonomic translation table (C. Arnold, Matthews and Nunn, 2010).

2.2.2 Statistical analysis

The multivariate structure of the data was explored through a phylogenetically controlled principal components analysis (hereafter pPCA). Previous well-known studies using PCA have failed to control for phylogenetic relationships (Finlay and Darlington, 1995; de Winter and Oxnard, 2001). The phylogenetic relatedness of species in a sample gives the data an underlying structure where more recently diverged taxa are likely to share more phenotypic similarities, which therefore renders them non-independent, violating the assumption of independent data (Harvey and Pagel, 1991). All data were log10 transformed prior to analysis to satisfy assumptions of normality. Lambda was estimated by maximum likelihood. The PCA was applied to a correlation matrix of standardised data using the package "phytools" (Revell, 2012) in R (R Development Core Team, 2015).

The size of brain structures varies with the overall size of the brain and body. Since this study seeks to test for mosaic change which is independent of these allometric effects, it is necessary to remove this variance so that change independent of size may be examined. Previous work has frequently used ratios or residuals to correct for size. There are a number of issues with these methods which make them inappropriate. The use of residuals as data can introduce bias (Garcia-Berthou, 2001; Freckleton, 2009), while ratios or proportions conflate independent change in structure sizes with allometric scaling effects (Albrecht, Gelvin and Hartman, 1995; Barton, 2002; Barton and Venditti, 2013). The arguably least controversial method of achieving size correction is to include a size variable in the PCA as a covariate. Analysis was therefore run in two conditions; one controlling for body size by including it as a covariate, and one with no size control to capture patterns of change that are not independent of size.

PCA extracts as many components as there are variables. A major issue with PCA is that the criteria for retention and interpretation of the components is to some extent subjective. Although the loadings characterise the component, it is down to the experimenter to ascribe meaning to it. There is also some disagreement over how to rationalise the number of components to retain. A commonly used method is Kaiser's Criterion, where a component is only retained if it has an eigenvalue of more than 1. A component with an eigenvalue of less than 1 indicates that it explains less variance than one of the original variables, and so does not exceed the explanatory power of the original observed variables. However, this criterion has been criticised as the variance accounted for by a component with a given eigenvalue varies with the number of variables; decreasing as more variables are added

(Field, Miles and Field, 2012). With this in mind, only those components which had a clear biological meaning were extracted in the current analysis.

When interpreting what each component might represent, it is necessary to determine a cut-off point for correlations between the original variables and the component. Below this cut-off the original variable is deemed not to make sufficient contribution to the component to be of explanatory utility. This study followed Tabachnick and Fidell's suggestion that component loadings of less than .32 should be disregarded as below this the original variable shares less than 10% variance with the component (Tabachnick and Fidell, 2012).

As an alternative way to examine potential differences in brain composition across clades, patterns of correlated evolution amongst the brain structures were analysed using PGLS. PGLS analysis was conducted in R (R Development Core Team, 2015) using the package "caper" (Orme *et al.*, 2013). Neocortex and cerebellum volume were used as outcome variables in two separate regressions, with each of the other 8 structures as predictors. Consistent with the PCA relative condition, body size was included as a predictor variable in order to control for its effect on brain structure size (Nunn, 2011). Since this chapter is exploratory, no explicit predictions are made, apart from a positive association between neocortex and cerebellum size in all three orders as these structures have previously been found to correlate in mammals (Barton, 2012).

2.3 Results

2.3.1 pPCA – Mammalia

All loadings on the first component in both size corrected and non-size corrected PCAs are negative and large (Table 2.3-1). This, coupled with the large percentage of variation it explains suggests that this component represents size. After the first component which accounts for the vast majority of the variance in the data (almost 93% in both conditions) and consequently has a very large eigenvalue, no other component's eigenvalue exceeds 1. However, as discussed in the Methods section above, Kaiser's criterion is not necessarily the most appropriate way of deciding how many components to extract. The second component has one large loading from the main olfactory bulb. The loadings from the rest of the structures are all very small, with only the neocortex and cerebellum reaching loadings above 0.1. The second component therefore has a fairly clear biological meaning, linked to the size of the main olfactory bulb. Components subsequent to the first two had low loadings,

explained very little variance, and had no obvious biological meaning. They were therefore disregarded.

The PCA results closely mirror those of Finlay and Darlington (1995), both in terms of the component loadings and the amount of variance explained by the components, despite the absence of phylogenetic correction in their analyses. The body size covariate condition and no size correction condition show near identical results. The bivariate plots (Figures 2:a & 2:b) show that the primates appear to be grouped in to a cluster which is quite distinct from the other mammals. Their position along both components contributed to this separation, suggesting that the primates are distinct from the other clades in terms of having overall larger brain structures and also smaller olfactory bulbs (both absolutely and relative to body size).

Table 2.3-1 - Mammal pPCA summary

		Covariate = body size		No size correction	
		PC1	PC2	PC1	PC2
Loadings	Body	-0.96	-0.04	-	-
	MOB	-0.87	0.49	-0.87	0.5
	Septum	-0.98	0.02	-0.98	0.02
	Striatum	-0.98	-0.05	-0.98	-0.05
	Amygdala	-0.96	-0.01	-0.97	-0.01
	Hippocampus	-0.96	-0.02	-0.96	-0.03
	Neocortex	-0.98	-0.14	-0.98	-0.14
	Cerebellum	-0.98	-0.13	-0.98	-0.13
	Medulla	-0.98	-0.08	-0.98	-0.08
Variance explained (%)		92.58	3.21	92.7	3.6
Eigenvalue		8.3	0.29	7.4	0.3

Phylogenetic principal components analysis applied to all three mammalian orders. Dashes signify instances where variable was not included in the model. MOB = main olfactory bulb

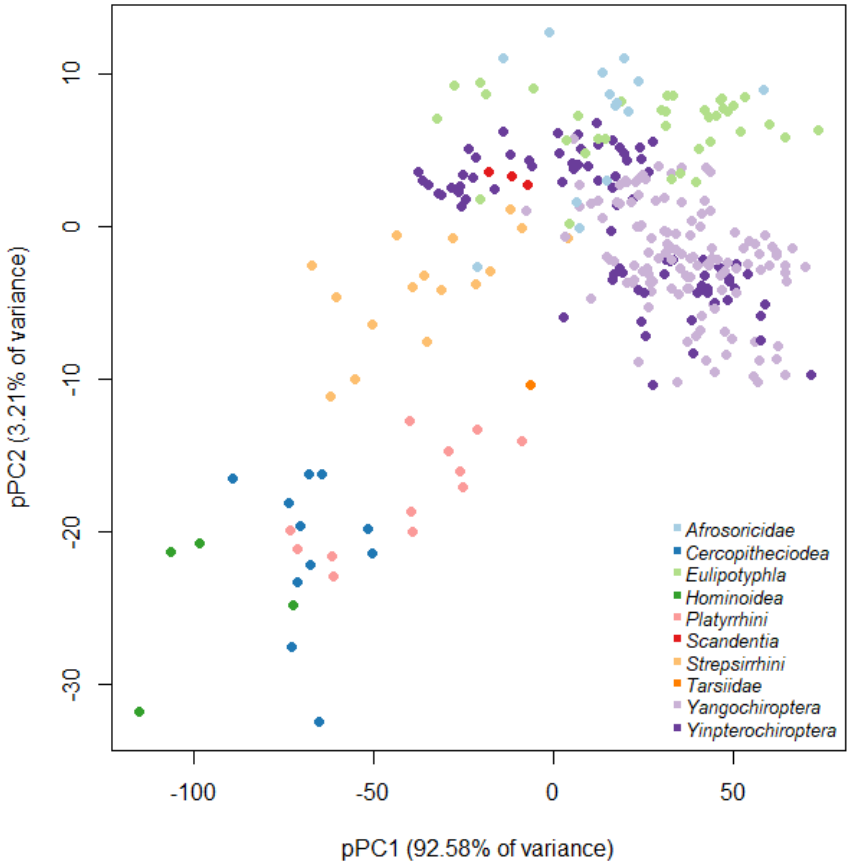


Figure 2:a – pPCA of Mammalian brain structures relative to body size

Phylogenetic principal components analysis applied to all three mammalian orders. Body size was included as a covariate to control for brain-body allometry.

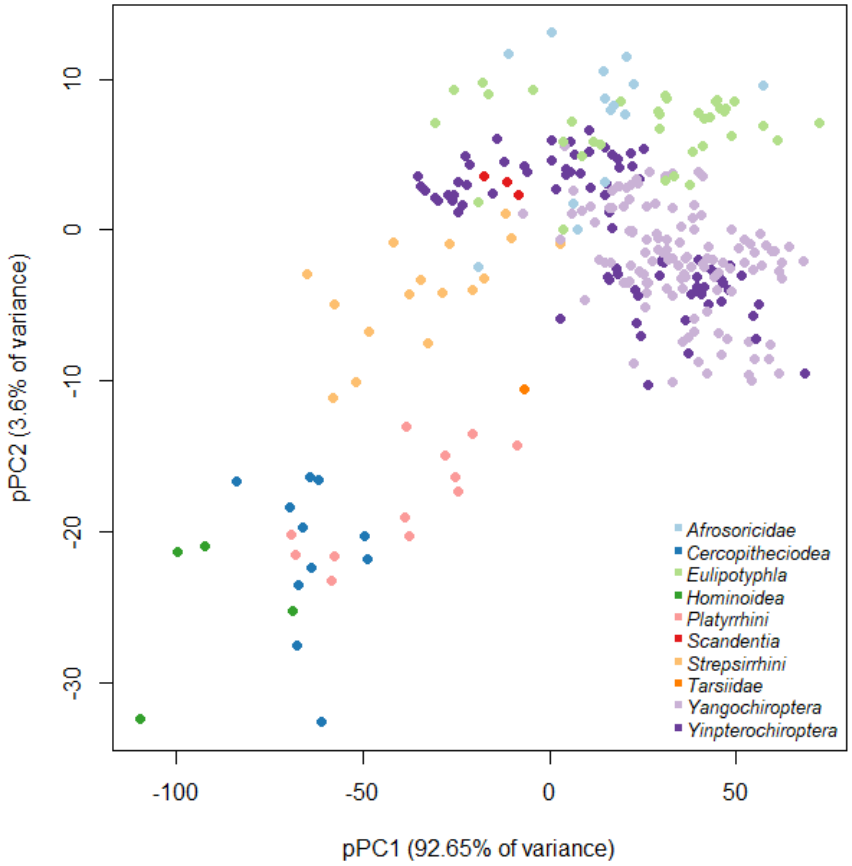


Figure 2:b - - pPCA of mammalian brain structures with no size correction

Phylogenetic principal components analysis applied to all three mammalian orders. Body size was not included in the model.

2.3.2 pPCA – Primates

As was found in the mammal-wide analysis, in the primates, after the very strong first component, no other component reaches the threshold of Kaiser's criterion (Table 2.3-2). Again, the loadings show a very strong first component on to which all structures load approximately equally, suggesting this component represents a general size factor. The second component is very heavily loaded by the main olfactory bulb. This second component represents around 7% of the variance in the primate sample, an increase on ~3.5% in the mammal-wide analysis. In both the primate and mammal-wide analyses, the olfactory bulb also loads substantially less heavily on to the first component than all the other structures. This suggests that variation in this structure is more independent of overall body or brain size than the other structures.

There is clear separation of the haplorhines and strepsirrhines along the second component in both conditions (Figures 2:c & 2:d). Apes are at the extreme of the first component which is unsurprising given this largely represents overall size. Strepsirrhines cluster more towards the positive end of component 2, while the haplorhines score negatively, reflecting their decreased dependence on olfaction and increased reliance on vision (Barton, Purvis and Harvey, 1995; Barton, 1998).

Table 2.3-2 - Primate pPCA summary

		Covariate = body size		No size correction	
		PC1	PC2	PC1	PC2
Loadings	Body	-0.95	0.06	-	-
	MOB	-0.66	0.75	-0.66	0.75
	Septum	-0.98	-0.08	-0.98	-0.07
	Striatum	-0.98	-0.08	-0.98	-0.07
	Amygdala	-0.97	-0.02	-0.97	-0.01
	Hippocampus	-0.98	-0.15	-0.96	-0.14
	Neocortex	-0.98	-0.13	-0.98	-0.12
	Cerebellum	-0.99	-0.08	-0.99	-0.08
	Medulla	-0.99	-0.02	-0.98	-0.01
Variance explained (%)		89.21	6.93	89.1	7.75
Eigenvalue		8.03	0.62	7.13	0.62

Phylogenetic principal components analysis applied to the order Primates. Dashes signify instances where variable was not included in the model. MOB = main olfactory bulb

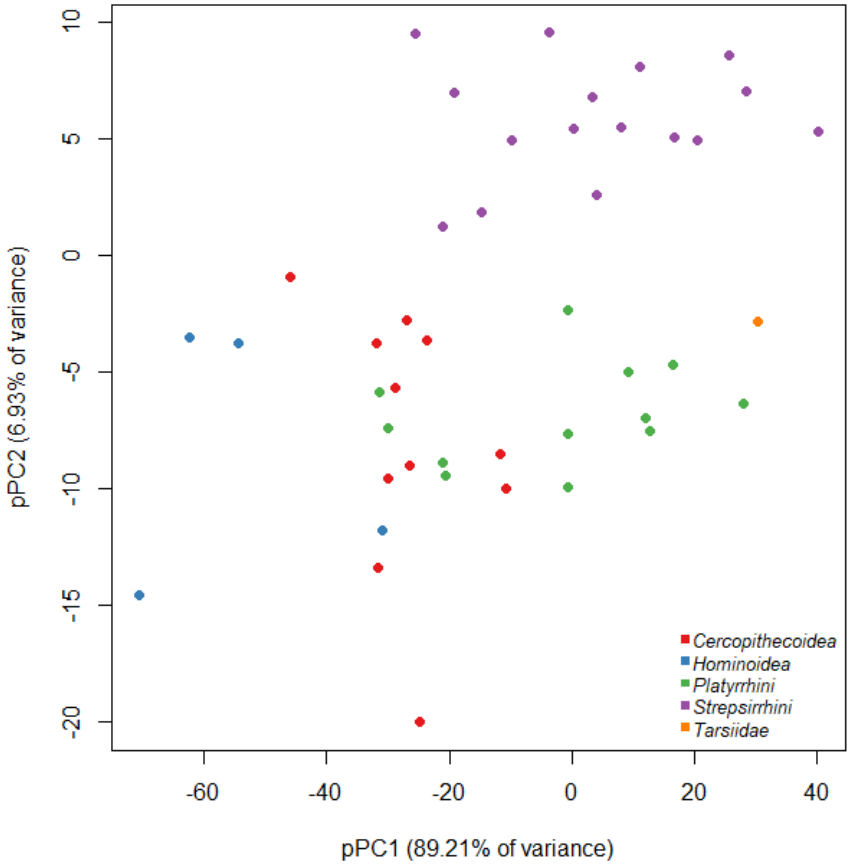


Figure 2:c – pPCA of primate brain structures relative to body size

Phylogenetic principal components analysis applied to the order Primates. Body size was included as a covariate to control for brain-body allometry.

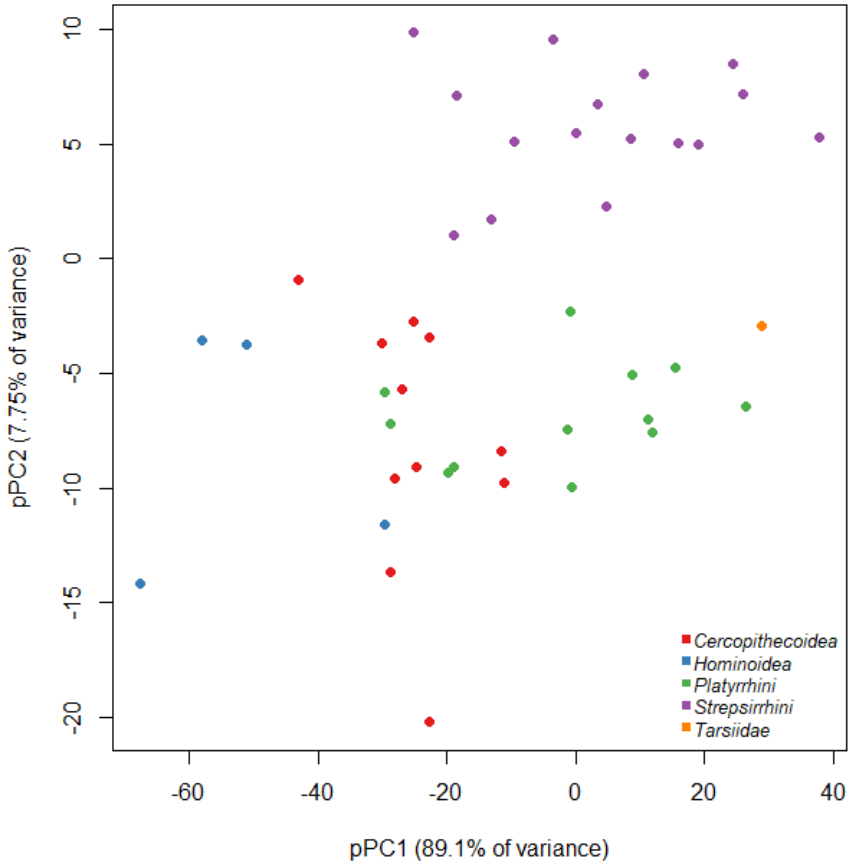


Figure 2:d – pPCA of primate brain structures with no size correction

Phylogenetic principal components analysis applied to the order Primates. Body size was not included in the model.

2.3.3 PGLS – associations between neocortex and cerebellum volume

In Mammalia, when body size and the other brain structures are controlled for, there is a strong positive partial correlation between the cerebellum (as the outcome variable) and neocortex volume (Table 2.3-4, phylogenetic least squares (PGLS); $\lambda = 0.98$, $t_{8,289} = 7.16$, $p < 0.0001$). There are also positive correlations between cerebellum volume (as the outcome variable) and the size of the medulla ($t_{8,289} = 6.8072$, $p < 0.0001$) and the hippocampus ($t_{8,289} = 2.4757$, $p < 0.05$).

Overall, the phylogenetic least squares regression shows quite different patterns of coevolution of brain structures between the three orders. The only relationship which is consistently evident across all three orders is that between neocortex and striatum volume. The correlation between the cerebellum (as outcome variable) and neocortex seen in the Mammalia analysis endures when primates ($\lambda = 1$, $t_{8,35} = 4.89$, $p < 0.0001$) and bats (($\lambda = .95$, $t_{8,192} = 4.49$, $p < 0.0001$) are analysed independently (Table 2.3-4). For the primates, the neocortex is the only brain structure whose size correlates with cerebellum size. Primates are also the only order whose cerebellum volume does not correlate with medulla volume (Table 2.3-4). In bats, both the medulla ($t_{8,192} = 6.29$, $p < 0.001$) and the hippocampus ($t_{8,192} = 3.37$, $p < 0.001$) are also correlated with cerebellum size. Cerebellum and neocortex volume are unrelated in insectivores ($\lambda = 1$, $t_{8,44} = 1.32$, $p > 0.05$). Instead cerebellum size is correlated with medulla ($t_{8,44} = 4.93$, $p < 0.001$) and striatum size ($t_{8,44} = 2.69$, $p < 0.05$). A notable difference between primates and the other two orders is their pattern of associations with the medulla. Primates exhibit a positive association between neocortex and medulla volume, which is absent in bats and insectivores (Table 2.3-3). Conversely, bats and insectivores exhibit a positive association between cerebellum and medulla volume, which is absent in primates (Table 2.3-4).

Table 2.3-3 – PGLS regression of neocortical volume on other brain structures

	Mammalia (n=298)	Primates (n=44)	Chiroptera (n=201)	"Insectivora" (n=53)
Predictor	t_{289} (p)	t_{35} (p)	t_{192} (p)	t_{44} (p)
(Intercept)	**4.5** ‡	**4.86** ‡	**3.66** ‡	**2.2** *
Cerebellum	**7.2** ‡	**4.27** ‡	**4.44** ‡	1.32 (0.19)
MOB	**-4.77** ‡	**-2.84** †	-0.92 (0.36)	**-3.51** †
Septum	0.17 (0.87)	**3.62** ‡	**-2.04** *	1.26 (0.21)
Striatum	**10.07** ‡	**3.57** †	**8.4** ‡	**4.13** ‡
Amygdala	**4.71** ‡	-0.14 (0.89)	**4.33** ‡	1.37 (0.18)
Hippocampus	0.05 (0.96)	**-3.68** ‡	1.64 (0.1)	0.72 (0.47)
Medulla	-0.4 (0.69)	**2.21** *	-0.33 (0.74)	0.16 (0.87)
Body size	0.97 (0.33)	-1.92 (0.06)	**2.72** †	0.32 (0.75)
λ	.97	.81	.90	1
r^2	.97	.99	.97	.97

Bold denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
* = p <0.05, † = p <0.01. ‡ = p <0.001

Table 2.3-4 – PGLS regression of cerebellar volume on other brain structures

	Mammalia (n=298)	Primates (n=44)	Chiroptera (n=201)	"Insectivora" (n=53)
Predictor	t_{289} (p)	t_{35} (p)	t_{192} (p)	t_{44} (p)
(Intercept)	-0.003	-1.24 (0.22)	1.07 (0.29)	-1.47
Neocortex	**7.16** ‡	**4.89** ‡	**4.49** ‡	1.32 (0.19)
MOB	-1.97 (0.05)	0.56 (0.58)	-0.69 (0.49)	-1.4 (0.17)
Septum	-0.12 (0.9)	-0.63 (0.54)	-1.09 (0.28)	0.15 (0.88)
Striatum	1.65 (0.1)	0.01 (0.99)	0.48 (0.64)	**2.69** *
Amygdala	0.92 (0.36)	1.72 (0.09)	0.39 (0.7)	-1.05 (0.3)
Hippocampus	**2.48** *	1.2 (0.24)	**3.37** ‡	1.89 (0.07)
Medulla	**6.81** ‡	-0.72 (0.48)	**6.29** ‡	**4.93** ‡
Body size	1.22 (0.22)	**2.07** *	1.36 (0.17)	**-2.04** *
λ	.98	1	.95	1
r^2	.97	.98	.96	.98

Bold denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
* = p <0.05, † = p <0.01. ‡ = p <0.001

2.4 Discussion

2.4.1 Evidence supporting concerted brain evolution

Overall size is undoubtedly a major element in the variation in brain composition across species. Across mammalian species, almost 93% of the variance in brain structure volumes is accounted for by the first principal component, representing overall size (whether this represents overall brain or body size is unclear as these cannot be distinguished in this analysis). The lack of any major differences in loadings or graphical patterns according to whether size is accounted for or not further demonstrates that the majority of the variance in brain composition in absolute and relative terms is size-linked. The pPCA results very closely resemble those of Finlay and Darlington (1995), despite the application of phylogenetic correction (Table 2.3-1). Kaiser's criterion (see "Methodology" above for problems with this threshold) would dictate that only the first principal component can be extracted, suggesting that there is no meaningful variation left over after accounting for size. Also, after the first two components which appear to describe a general size factor and olfactory bulb, the loadings of the original variables are very low and show relatively little variation. This stands in contrast to what was predicted if brains evolved in a mosaic fashion, where structures which have undergone correlated evolution were expected to load on to common components. One possible interpretation of the pPCA results therefore could be a concerted evolution perspective which holds size (and the presumed developmental constraints linked to it) to be the significant factor in brain composition and that any selection for function causes an increase in overall brain size rather than an increase in a specific structure or network that mediates said function (Finlay and Darlington, 1995).

This separation of the main olfactory bulb (MOB) from the rest of the structures in the PCA also echoes previous findings. Finlay and Darlington found that this structure had a much lower correlation with overall brain size than any of the others. The lower loading of the main olfactory bulb on to the first component seems to suggest a pattern of size variation in this structure which has some independence relative to the others examined. This relative independence of the size of the olfactory system from that of the rest of the brain has been reported in a number of studies (Finlay and Darlington, 1995; Yopak *et al.*, 2010; Smaers and Soligo, 2013), but the reason underlying its independence are not always clearly articulated. It has been suggested, in anthropoid primates at least, that the olfactory system has been

relatively conserved during the course of the clade's evolution, and so the size of the rest of the brain has been comparatively much more variable (Smaers and Soligo, 2013).

When comparing the results of the present study with a previous PCA study in mammals which found support for a mosaic pattern of brain structure variation, that of de Winter and Oxnard (2001), there is clearly major divergence. Where they were able to extract a number of components with clear biological meaning which split taxa by ecological niche, the current study found no such components. Accounting for phylogenetic relatedness by using phylogenetic comparative statistical methods has contributed to this discrepancy, but more significant is the manner in which each study deals with allometry. As mentioned in the introduction, since de Winter and Oxnard use proportional measures of one structure relative to another, they conflate independent change in structures with allometric scaling. Therefore, change in the volume of a structure that occurs independently of the rest of the brain cannot be distinguished from change that occurs due to overall brain size change. Accounting for allometry in the study of comparative brain composition variation is complex. Firstly, grade shifts in relative structure sizes can obscure the true nature of their allometric scaling (Nunn and Barton, 2001). Barton and Harvey (2000) reported that without the confounding effect of such shifts, the neocortex scaled near isometrically with brain size. Without accounting for grade shifts, the variation in the size of this structure had been attributed entirely to a single universal mammalian allometric trajectory (Finlay and Darlington, 1995). Second, the relationship of brain structures to overall brain size, and of brain size to body size is also variable (Montgomery *et al.*, 2013). Since these are the most commonly used metrics for controlling for size, this variability presents a problem for comparison across clades. This has led some researchers to prefer structures whose relationship with both brain and body size is thought to be highly conserved, like the medulla (de Winter and Oxnard, 2001) or spinal cord (Willemet, 2013).

2.4.2 Evidence supporting mosaic brain evolution

While the PCA loadings appear to support a concerted brain evolution pattern, the separation of the taxa in the PCAs (Figures 2:a – 2:d) and different patterns of correlated evolution between structures in the PGLS indicate that different patterns of brain composition evolution exist across taxa. The PGLS results show that these taxa have independent patterns of relationships between structures, suggesting that they have undergone different selection pressures which have shaped their brains in different ways (Tables 2.3-3 & 2.3-4). This stands in contrast to the concerted/linked regularities hypothesis which would have seen all 3

orders following the same pattern, stretching out along a single component related to size. Therefore, these results do not support a universal mammalian pattern of brain evolution in these taxa. Additionally, in both the mammal-wide and primate only PCA analyses, the second component clearly differentiates the Haplorhini from the other taxa, including the Strepsirrhini. The clustering of the haplorhines towards the negative end of the second component and the heavy loading of the main olfactory bulb on to this component reflects the diminished role of olfaction in this taxon (Barton, 2006b).

The results of the PGLS analysis are also in clear agreement with previous work which has demonstrated that the volume of the cerebellum and neocortex are correlated both in primates and mammals at large (Whiting and Barton, 2003; Smaers, Steele and Zilles, 2011; Barton, 2012). The relationship is apparent in the full analysis incorporating all 3 orders and also in individual analyses of the primates and bats. However, the relationship does not hold for the "insectivores". As mentioned above, this grouping in paraphyletic and so it could be misleading to analyse these species together in an analysis like this. However, this grouping was necessary to facilitate clear comparison with previous analyses which have used it.

2.4.3 A false dichotomy?

Taken together, the pPCA and PGLS results ultimately support the consensus in the literature; both concerted and mosaic influences combine to change brain composition (Barton, 2006a; Herculano-Houzel, Manger and Kaas, 2014; Hoops *et al.*, 2017; O'Donnell *et al.*, 2018). Both processes have been observed in individual taxa (Gutiérrez-Ibáñez *et al.*, 2014; Herculano-Houzel, Manger and Kaas, 2014; Hoops *et al.*, 2017). There is no doubt that development and evolution are linked (Montgomery, Mundy and Barton, 2016). Rather, there is disagreement about the relative contribution of each to the variation in comparative brain composition (Striedter, 2006). Proponents of concerted evolution maintain that mosaic evolution is rare (Striedter, 2006), but have suggested developmental mechanisms under which it can occur. They suggest that selection may act directly upon developmental scheduling mechanisms (Charvet and Finlay, 2012), but this only helps us understand the mechanics of mosaic changes, not whether or not mosaic evolution is a major force shaping brain composition. Some propose that allometric constraints dominate until selection forces a change, so that mosaic changes can overcome constraints in an otherwise concerted brain when the species is under direct selective pressure (O'Donnell *et al.*, 2018). The relative dominance of one model over the other may even be clade specific. For example, Hoops et al.

(2017) suggests that brain evolution in the cartilaginous and bony fishes exhibits mostly concerted and mostly mosaic patterns respectively.

As detailed above, mosaic brain evolution has been observed and experimentally induced in a wide range of taxa. Genetic evidence is also supportive of a significant role of mosaic evolution in brain composition, with developmental constraints, while present, not a strong factor (Hager *et al.*, 2012; Harrison and Montgomery, 2017). The PGLS results above support the predictions of a mosaic model; showing specific correlations between structures which are independent of overall size and reflective of connectivity. If the evidence for mosaic change is so strong, then why does the pPCA recover no evidence of it? It is possible that patterns of mosaic evolution are not detected by an approach like PCA for the following reasons. A principal component analysis necessarily finds axes of variation which are not related to each other (i.e. are orthogonal to one another). For coevolved groups of structures to be represented by individual components, groups of structures which have coevolved would need to be uncorrelated with other coevolved systems. Given the distributed nature of functions across groups of structures and the resulting non-independence of these groups, one might expect that no one structure or group of structures would load heavily on to a component. In addition, the data analysed here are based on traditional grossly anatomically defined structures which do not represent functional systems very well, as distributed systems cut across these gross structures (Buckner and Krienen, 2013). Dividing the brain in to these anatomical structures may therefore not reflect biological reality in terms of how selective pressures influence the relative sizes of structures.

2.4.4 Setting the scene

The pPCA analysis shows a clear separation of the primates from the other orders. This separation occurs along the two major axes of variation in brain composition in the mammalian taxa analysed – namely: overall size and the size of the olfactory bulb. The primates therefore constitute an important and appropriate taxon for the key questions in brain evolution. This order has been the subject of intense research interest and so a large amount of detailed neurovolumetric, ecological, behavioural and developmental data are available. The inclusion of the other mammalian taxa discussed in this chapter is largely prohibited by both availability of data and the scope of the project. The following chapters investigate the mosaicism in brain evolution discussed in this chapter, focusing on the order Primates.

3 Re-evaluating the link between brain size and behavioural ecology in primates

3.1 Introduction

Absolute brain size varies almost a thousand-fold across the order Primates (Barton, 2012), and the adaptive significance of this variation has been the subject of intense interest. As neural tissue imposes costs (Aiello and Wheeler, 1995), evolutionary increases in brain size are assumed to confer benefits in terms of enhanced cognitive abilities (Healy and Rowe, 2007; Weisbecker *et al.*, 2015). Although this assumption has received support from studies demonstrating positive associations between brain size and cognitive performance (MacLean *et al.*, 2012, 2014, Kotrschal *et al.*, 2013, 2015; Benson-Amram *et al.*, 2016), the selection pressures responsible are still poorly understood.

3.1.1 Ecological or social brains?

A classic approach to this problem is to examine which specific aspects of lifestyle correlate with brain size across species. In primates, two broad categories of hypothesis have been tested in this way; ecological and social. Ecological hypotheses mainly relate to the foraging demands of a species' ecological niche (Harvey and Rambaut, 2000; Barton, 2006c; Mars *et al.*, 2014; Parker, 2015). Effects of diet (Clutton-Brock and Harvey, 1980; Harvey, Clutton-Brock and Mace, 1980; Fish and Lockwood, 2003; Walker *et al.*, 2006; van Woerden, van Schaik and Isler, 2010; Swanson *et al.*, 2012; DeCasien *et al.*, 2017), home range size (Milton and May, 1976; Clutton-Brock and Harvey, 1980; Parker, 2015), terrestriality (Sawaguchi, 1990) and activity period (Barton, Purvis and Harvey, 1995; Barton, 1996) on brain or brain component size have been reported, and explanations for such effects invoke a range of information-processing capacities, including spatial or spatio-temporal memory and visual processing (Clutton-Brock and Harvey, 1980; Milton, 1988; Barton, Purvis and Harvey, 1995; Barton, 1998). In contrast, the Social Brain Hypothesis (SBH) proposes that the principal selection pressure responsible for variation in primate brain size is the cognitive demands of managing social relationships within bonded groups (Jolly, 1966; Humphrey, 1976; Whiten and Byrne, 1988; Dunbar, 1992; Barton and Dunbar, 1997; Dunbar and Shultz, 2007a), a hypothesis that has received considerable empirical support (Dunbar, 1992; Barton and Dunbar, 1997; Dunbar and Shultz, 2007a). Relationships between sociality and brain size

have also been reported in other mammalian taxa such as Ungulates (Shultz and Dunbar, 2006, 2007), Cetacea (Fox, Muthukrishna and Shultz, 2017)and Carnivora (Pérez-Barbería, Shultz and Dunbar, 2007; Shultz and Dunbar, 2007; Swanson *et al.*, 2012; Holekamp *et al.*, 2015).

3.1.2 Questioning the Social Brain

Nevertheless, some studies have failed to find a statistical link between brain size and sociality (Clutton-Brock and Harvey, 1980; Swanson *et al.*, 2012; Holekamp *et al.*, 2015; DeCasien *et al.*, 2017), and apparent exceptions, in terms of large-brained but not conspicuously social taxa, suggest that factors other than sociality may have been influential (Byrne, 2006; Swanson *et al.*, 2012; van Schaik *et al.*, 2012). In particular, a recent analysis by DeCasien et al. (2017) found that diet, and not social group size, correlates with brain size in primates. DeCasien et al. point to several possible explanations for the correlation with diet that invoke the cognitive basis of foraging skills. Shultz & Dunbar (2007) had earlier acknowledged that primate brain size correlates with diet but argued (a) that this reflects energetic constraints on brain size rather than selection on foraging skills, and (b) that brain size correlates with sociality independently of diet. The regression models supporting the latter conclusion were based on relatively small sample sizes, and, using a larger sample size, DeCasien et al. (2017) failed to find an independent effect of social group size after accounting for body size and diet, as well as for phylogenetic uncertainty. On the other hand, Shultz and Dunbar (2007) incorporated a wider range of ecological variables into their model. Here we combine the strengths of these studies and evaluate the possible effects of their use of different data sets; that is, we use phylogenetic comparative analysis applied to large sample sizes, we incorporate all the key behavioural-ecological predictors examined in previous studies, and we account for phylogenetic uncertainty. Error variance in predictors theoretically has a major impact on the results of regression analyses and is likely to be considerable in the case of behavioural measures collated from field studies conducted by different researchers using different methods on different populations. However, almost nothing is known about the effects of this problem on determining the behavioural correlates of brain size. A novel feature of our study is therefore that we assess the robustness of results by replicating analyses across datasets. A lack of such robustness would have significant implications for attempts to infer selection pressures from analyses that neglect this issue.

3.2 Materials and Methods

3.2.1 Data sources

Brain size (endocranial volume) and body mass were obtained from previously published compilations (Isler *et al.*, 2008; van Woerden, van Schaik and Isler, 2010, 2014; van Woerden *et al.*, 2012). Whilst it might be argued that the SBH specifically invokes the neocortex as the relevant brain structure (Barton and Dunbar, 1997; Shultz and Dunbar, 2006; Dunbar and Shultz, 2007a), proponents of the SBH refer to the hypothesis as an explanation for brain size and have used both overall brain and neocortex size (Shultz and Dunbar, 2006, 2010) arguing that brain size and neocortex size are closely related, because the neocortex comprises a large proportion of whole brain volume (Shultz and Dunbar, 2007; Dunbar and Shultz, 2017). Using brain size markedly increases sample sizes and statistical power. Nevertheless, we recognise that these two measures could theoretically give different results (see 3.4 Discussion).

Two datasets on primate behavioural ecology were analysed. The first (hereafter referred to as 'dataset 1') is a previously unpublished dataset compiled from the literature by Isler, providing updated, high quality data on primate behavioural ecology; favouring wild samples over captive, larger samples over smaller, original contributions over compilations, and more recent sources over older ones (Isler *et al.*, 2008; van Woerden, van Schaik and Isler, 2010, 2014; van Woerden *et al.*, 2012). For sexually dimorphic species (size difference > 10%), female values for endocranial volume (hereafter "ECV") and body mass were used. For all other species, means were calculated across males and females. If available, body mass was taken from the same specimens as ECV. Otherwise, the largest available sample of wild body mass data was used. Dataset 1 includes information on diet composition (the percentage of time spent feeding on different dietary items), size of sleeping groups and of foraging groups, day ranges, and home range sizes. Dataset 2 was compiled from the literature by Nunn and van Schaik (2002). It provides values for female body mass, activity period, substrate use, and diet. As body size in dataset 2 is derived only from female specimens, for comparability we also ran an analysis on dataset 1 using only female body size estimates (Appendix 13). Datasets 1 and 2 are not independent, as their sources overlap. Therefore, in order to test for robustness of results across strictly independent datasets, we also created subsets of the data by randomly selecting different species from each original dataset.

3.2.2 Selection of ecological variables

Five behavioural-ecological variables were selected for analysis, based on the previous literature (Milton and May, 1976; Clutton-Brock and Harvey, 1980; Milton, 1988; Dunbar, 1992; Dunbar, 1998; Barton, 1999; Dunbar and Shultz, 2007a): two continuous variables (home range size (ha) and social group size) and three dichotomous categorical variables: activity period (nocturnal/diurnal), substrate use (terrestrial/arboreal) and diet (folivore/non folivore). Rather than presenting quantitative estimates, Nunn and van Schaik (2002) classified species' diet categories based on the food type that occupied the largest proportion of feeding time. We therefore used the same criterion to categorise diet in dataset 2. However, diet is subject to marked intraspecific variation in relation to seasonal and local differences in the relative abundance of different food types (Melin *et al.*, 2014). Hence, categorising species' diet according to percentage of feeding time can create anomalies, in which closely related species with similar foraging niches are placed in different categories due simply to the quantitative estimates being based on insufficient or inaccurate samples. We therefore ran an additional separate analysis for dataset 1 in which folivores were more strictly defined as only those species with clear physiological specialisations for folivory (Appendices 19 & 20) (Hladik, 1978; Chivers and Hladik, 1980). As in previous analyses (Barton, Purvis and Harvey, 1995; Barton, 1996, 2006c), diurnal species were defined as those that regularly forage and are active during the day, therefore including the few cathemeral lemurs which are more diurnal than their strictly nocturnal close relatives (Griffin, Matthews and Nunn, 2012; Donati *et al.*, 2013).

3.2.3 Selection of group size data

Dataset 2 (Nunn and van Schaik, 2002) provides both 'population group size' and 'foraging group size'. The authors define population group size as "…the animals that come together frequently, usually to sleep together and among which foraging units have highly overlapping ranges." (p. 202), whereas foraging group sizes include the smaller, temporary parties or subgroups that form in response to immediate daily foraging conditions. Since the SBH relates to communities of individuals that associate habitually, we used population group size from Dataset 2. Dataset 1 (Isler, no date) recorded both sleeping and foraging group size. A third group size measure ("Combi Group Size") takes the largest of the sleeping and foraging group figures. Combi Group Size therefore reflects the number of individuals who regularly associate and is thus essentially definitionally the same as population group size from Dataset 2. We therefore used Combi Group Size in our primary analyses of dataset 1. However, we

also reran the analyses with sleeping group size only (where available) and found no qualitative difference in results (Appendix 12). While group size may be a relatively indirect measure of primate social complexity (Dunbar, 1998; Fischer *et al.*, 2017), it is the one that forms the foundation of work on the SBH (Dunbar, 1998; Dunbar and Shultz, 2007a), and as we intended to revisit the conclusions of that work it is necessary to use the same metrics as used in those papers.

3.2.4 Statistical analysis

Both analyses used the same endocranial volume data; only the behavioural-ecological data differed. We used phylogenetic generalised least squares regression (PGLS) to analyse the correlated evolution of the five behavioural-ecological variables and endocranial volume. Data were analysed in the R (R Development Core Team, 2015) packages "ape" (Paradis, Claude and Strimmer, 2004), "picante" (Kembel *et al.*, 2010), "caper" (Orme *et al.*, 2013) and "nlme" (Pinheiro *et al.*, 2015). Pagel's ʎ (Pagel, 1999) is a scaling parameter, used to scale the variance co-variance matrix according to the expected variance given a phylogenetic tree, thus accounting for the confounding effect of phylogenetic relatedness in comparative studies (Nunn, 2011). ʎ was estimated by maximum likelihood. For the PGLS analyses, the phylogeny used was the consensus tree incorporating branch length estimates from the 10k Trees project (Arnold, Matthews and Nunn, 2010). Body mass was included as a covariate in the regression to control for its effects on endocranial volume following Freckleton (2002), Smith (1999), and Garcia-Berthou (2001). This method of body size correction is preferred over analysis of residuals as it avoids biased parameter estimates (Freckleton, 2002). Including body mass as a covariate also has the benefit of controlling for any effects of body mass on other predictors, which is likely to be a particular issue for home range size. The granularity of the environment as perceived by the animal is likely to be dependent upon its size. For example, an increase of 1 hectare would likely have very different implications for a 50g mouse lemur than for an 85kg gorilla.

All continuous variables (endocranial volume, body mass, group size, and home range size) were log10 transformed prior to analysis to satisfy the assumption of normality. Prior to the analysis, we inspected the distribution of the response and predictor variables and found them to be approximately symmetrically distributed. We inspected diagnostic plots for the model and found no evidence of violation of the assumptions of normality or homogeneity of residuals (Quinn and Keough, 2002). Models were checked for outliers with a studentised residual with an absolute value >3 (Field, Miles and Field, 2012). None were found. We

checked for collinearity between predictors in our models. Although statistically significant partial correlations were present for all predictors, none were above 0.67. Absolute correlations of less than .8 are deemed not to represent significant collinearity issues (Garland, 2012). Variance inflation factors (VIFs) (Quinn and Keough, 2002) were less than 1.4 in all cases which further reassured us that collinearity was not a significant problem in this case (Mundry, 2014).

3.2.5 Model comparisons

To assess the fit of the PGLS models, we constructed models which varied in complexity; from an allometric model in which body size was the sole predictor, models including body size and each predictor alone, and then added parameters to the model according to their p value (low to high). We then compared the AIC (Akaike's Information Criterion) (Akaike, 1974) for each model using the native "AIC" function in R (R Development Core Team, 2015). The AIC takes in to account the size of the sample and the number of predictors; penalising complex, over-paramaterised models (Quinn and Keough, 2002). Lower values of the AIC indicate better fitting, more parsimonious models. We also used log likelihood ratio tests to assess fit (Burnham and Anderson, 2002), run using the "lrtest" function in the lmtest package (Zeileis and Hothorn, 2002) in R (R Development Core Team, 2015).

3.2.6 Accounting for phylogenetic uncertainty

The PGLS analyses are based on a single consensus tree of the primates, but phylogenetic relationships are not known with certainty. To account for this issue and to additionally test whether this potential source of error in comparative studies has a significant impact on identifying correlates of brain size, we performed Bayesian phylogenetic regressions (Pagel, 1999) accounting for shared ancestry by integrating over a posterior sample of 1000 primate phylogenetic trees taken from the 10k trees project website (Arnold, Matthews and Nunn, 2010). We conducted these analyses using BayesTraitsV3 (Pagel and Meade, 2016). To account for the level of phylogenetic signal in our data we estimated the tree scaling parameter ʎ (Pagel and Meade, 2016). We used a uniform prior of -100 to 100 for all regression coefficients and a uniform prior of 0 to 1 for ʎ. We ran the analyses for 1,010,000 iterations, sampling every 1000 iterations removing the first 10,000 iterations as burn-in. To determine the significance of our regression coefficients we used pMCMC values which can be interpreted in a similar way to frequentist p-values (Hadfield, 2010).

3.3 Results

3.3.1 PGLS

Table 3.3-1 - Phylogenetic Least Squares (PGLS) regressions examining the effects of five behavioural-ecological variables on endocranial volume.

	Dataset 1 (n=144)		Dataset 2 (n=104)	
Predictor	t_{137}	p	t_{97}	p
Intercept	-5.5	**<0.001‡**	11.3	**<0.001‡**
Body Size	18.6	**<0.001‡**	13.3	**<0.001‡**
Activity period	2.5	**<0.05***	1.9	0.06
Terrestriality	0.4	0.69	-0.3	0.8
Folivory	-1.7	0.08	0.1	0.9
Group Size	1.7	0.1	0.1	0.9
Home Range Size	2.4	**<0.05***	2.8	**<0.01†**
Model summary:				
ʎ	.988		.997	
R2	.8		.75	

Predictor variable = endocranial volume. **Bold** denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
***** = p <0.05, **†** = p <0.01. **‡** = p <0.001

Table 3.3-1 presents the results of PGLS analyses on the two full datasets. In all cases ʎ was close to 1, indicating that the data are consistent with a Brownian motion model of trait evolution (Barton and Venditti, 2014). A simple allometric model regressing endocranial volume on body size alone explained 77% of the variation in dataset 1 and 73% in dataset 2. The full model (comprising all five behavioural-ecological variables) was highly significant in both dataset 1 (ʎ=0.99, r2=0.8, p <0.0001) and dataset 2 (ʎ=1, r2=.75, p <0.0001).

In dataset 1 home range size and activity period were both associated with endocranial volume after accounting for the effects of body size (positive associations between brain size and HRS and diurnality respectively) (ʎ=0.99, t6,108=2.1, p <0.05). The model based on dataset 2 (Isler, no date) also showed a significant positive partial correlation with home range size, (ʎ=0.99, t6,97=2.8, p <0.01), but the partial correlations with activity period did not reach significance (p=0.06), and no other behavioural-ecological variables were significantly correlated with brain size while accounting for these effects.

Table 3.3-2 - Phylogenetic Least Squares (PGLS) regressions examining the effects of five behavioural-ecological variables on endocranial volume with datasets matched for species.

	Dataset 1 (n=99)		Dataset 2 (n=99)	
Predictor	t_{92}	*p*	t_{92}	*p*
Intercept	-5.8	**<0.001‡**	11	**<0.001‡**
Body Size	16.9	**<0.001‡**	13	**<0.001‡**
Activity period	1.8	0.1	1.9	0.1
Terrestriality	0.3	0.8	-0.2	0.8
Folivory	-2.2	**<0.05***	0.1	0.9
Group Size	1	0.3	0.1	0.9
Home Range Size	1.3	0.2	2.5	**<0.05***
Model summary:				
Λ	.99		1	
R^2	.81		.75	

Predictor variable = endocranial volume. **Bold** denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
***** = p <0.05, **†** = p <0.01. **‡** = p <0.001

When each dataset was matched to include the same species and the same endocranial volume data, results changed, and again differed between datasets. Table 3.3-2 indicates significant partial correlations for diet in dataset 1 and for home range size in dataset 2. In both cases, the effect of activity period was now non-significant.

We next performed PGLS analyses on the datasets (i) after they had been made completely independent from each other, and (ii) after they had been reduced to include only species that appeared in Stephan et al.'s 1981 brain component volumes dataset (Stephan, Frahm and Baron, 1981). Again, results differed between the datasets and from the results reported above (see Appendices 4 & 9 for full results). Folivory showed a significant negative association with brain size in independent dataset 1, whereas there were no significant predictors after accounting for body mass in independent dataset 2. Similarly, no significant associations were found in the full multiple regressions on either dataset when they were matched to the Stephan et al. (Stephan, Frahm and Baron, 1981) species list. However, because the sample sizes in these analyses were small relative to the number of predictors, we used model comparisons to determine which combinations of predictors are best supported (see below).

3.3.2 Model Comparison

To establish which combination of variables model endocranial volume best in each dataset, we employed a model comparison approach using Akaike's Information Criterion (Akaike, 1974) and log likelihood ratio tests (Burnham and Anderson, 2002). We first subjected the full datasets to model comparison (Appendices 2 & 3).

AIC values indicate that the model offering the best and most parsimonious explanation of dataset 1 was one which included activity period, home range size, diet and group size. (model ix, Appendix 2). Following Burnham and Anderson (2002), an AIC difference (Δi) of less than 2 was considered to indicate substantial empirical support (p. 70). The best model was therefore not a significantly better fit to the data than models vii, viii and x (Δi < 2). AIC differences between the models fitted to dataset 2 (Table S3) showed that a model containing home range size and activity period was the best fit to the data, but model vi which included only body size (the covariate) and home range size provided a comparable fit (Δi < 2). Model viii (home range size, activity period and terrestriality) also gave a comparable fit according to the Δi < 2 rule, but a log likelihood ratio test showed that this addition of terrestriality did not significantly improve the fit (Appendix 3). In summary, these results show that endocranial volume is best modelled by different combinations of variables in the two datasets. Home Range Size was consistently present in the best models (Δi < 2) across the two datasets, appearing in all seven of the best models. Group size appeared in only two of the seven best models and only when accompanied by home range size, folivory and activity period.

As described above, the inclusion of different species in each dataset may result in the composition of the best models varying between datasets. We therefore also subjected the species matched datasets to model comparison, as detailed in Appendices 5 & 6.

The model comparisons for the species matched datasets show broad agreement with those of the non-matched, full datasets in Appendices 2 & 3. The best models still consistently included home range size, appearing in every model with substantial support (i.e. where Δi < 2) save one (model viii, Appendix 5). Group size appeared in only one of the best models, again together with home range size, folivory and activity period.

PGLS model comparisons for the Stephan et al. (1981) sample of species identified social group size as a significant predictor: in both datasets, group size and folivory were included

in the best model. The addition of home range size was found not to improve the fit in either dataset (Appendices 10 & 11).

3.3.3 Accounting for phylogenetic uncertainty

A Bayesian phylogenetic regression of the full datasets replicated the qualitative results of the PGLS analyses. In dataset 1, Home range size (posterior mean = 0.0247, 95%CI = 0.0241 to 0.0253, pMCMC=0.0066) and activity period (posterior mean=0.1327, 95%CI = 0.1293 to 0.262, pMCMC=0.0154) both had pMCMC values of less than 0.05 (Appendix 14), indicating that these traits were well supported (Pagel and Meade, 2016). Home range size was the only predictor with strong support in dataset 2 (posterior mean=0.0426, 95%CI = 0.0416 to 0.0436, pMCMC = 0.0007, Appendix 17). Appendices 15, 16 and 18 show the posterior distributions of estimates of those traits that had pMCMC < 0.05.

3.4 Discussion

We have re-examined the correlates of brain size in primates, using two large comparative datasets, and incorporating multiple potentially relevant behavioural variables within phylogenetic statistical models. Our results indicate that, even holding constant statistical methods, phylogeny, set of predictor variables, response variable data, and species sample, the behavioural and ecological correlates of brain size are sensitive to the use of different predictor datasets. Accounting for phylogenetic uncertainty did not affect this outcome.

3.4.1 Support for the Ecological Brain

This lack of robustness raises doubts about inferences from behavioural-ecological correlates of brain size based on analyses of single datasets and may help to explain divergent results between studies. To the extent that we find stability, there is stronger evidence for correlations with ecological factors, notably home range size, than for social group size, as found in Clutton-Brock and Harvey's pioneering study (Clutton-Brock and Harvey, 1980). Our results are also broadly in line with the more recent study of DeCasien et al. (2017), in finding stronger and more robust associations with ecological factors related to foraging than with social group size. However, our inclusion of additional variables and datasets also reveals differences. DeCasien et al. identified frugivorous diets as the key correlate of large brain size but did not examine home range size. In contrast, we found home range size rather than diet to be the most consistent correlate of brain size, but note that this varied between

datasets, suggesting their effects are hard to separate, perhaps because diet and ranging together form an adaptive 'syndrome': more frugivorous and (less folivorous) diets are strongly associated with more patchily distributed resources and larger home ranges (Nunn and van Schaik, 2002). The manner in which diet is categorised also appears to have an impact; when only species with biological adaptations to leaf processing are classified as folivorous, diet additionally becomes a significant predictor of brain size (Appendices 19 & 20). We also found some evidence for an association between activity period and large brain size, though this effect was small and variable across datasets, the potential reasons for which we discuss below.

3.4.2 Social Brain Hypothesis is not well supported

Evidence for a correlation between brain size and social group size after accounting for effects of other variables was weak. We found that this well-known correlation appears largely dependent on the particular sample of species in the Stephan dataset (Stephan, Frahm and Baron, 1981). One elaboration of the Social Brain Hypothesis accounts for dietary correlates of brain size in primates as a reflection of energetic constraints (Dunbar and Shultz, 2007a; Shultz and Dunbar, 2007; Dunbar and Shultz, 2017) . In this view, sociality selects for bigger brains and diet must become more frugivorous to provide the additional energy required to meet the costs. However, this hypothesis would presumably predict stronger correlations with diet than with home range size, which we do not find. In addition, we do not find support for the claim that social group size and brain size are robustly correlated after accounting for the effects of ecological variables (Shultz and Dunbar, 2007; Dunbar and Shultz, 2017). We agree with Dunbar & Shultz (Dunbar and Shultz, 2017) that, in principle, comparative analysis should differentiate between selection pressures and constraints, but it remains unclear how this can be achieved in practice. While path analysis has been suggested as a possible solution (Dunbar and Shultz, 2007a; Dunbar and Shultz, 2017), it is essentially a protocol for arranging a set of regression coefficients according to some causal hypotheses; it cannot be used to discover causality from correlational data (Denis and Legerski, 2003), it cannot solve the problem of instability across datasets, and it is as vulnerable to underlying issues with the data as are the regression analyses on which it is based. In summary, while it remains plausible that sociality is related to cognitive evolution in primates, we suggest that this can no longer be claimed on the basis of a strong or robust correlation between brain size and group size that remains after controlling for other variables.

3.4.3 Sources of instability

Why are results unstable, and what implications does this have for using them to infer selection on cognitive abilities? We highlight three empirical issues (data quality, statistical power and intrinsic intra-specific variability) as well as theoretical difficulties with brain size as a global measure of cognitive capacities. Data quality and replicability are major issues for comparative studies because of the diversity of sources and of the methods used by different researchers to collect the primary data (Borries *et al.*, 2013, 2016; Patterson *et al.*, 2014). Furthermore, many behaviours vary extensively within and between populations of the same species, and comparative studies routinely collapse this intra-specific variation into species-specific means. The validity of these mean values depends on the extent to which the variation has been sampled to a comparable extent across species, and on the assumption that inter-specific variation is substantial by comparison. For example, group size in different populations of terrestrial or semi-terrestrial cercopithecine species varies widely, depending on habitat, reflecting facultative adjustment of behaviour to local ecological conditions. Group size in yellow baboons (*Papio cynocephalus*) was found to vary between 8 and 44 within one study population (Stacey, 1986); the contrasts between Papio populations or sub-species is even more marked, with estimates of group size varying approximately 20-fold (Dunbar, 1992) and of home range size approximately 100-fold (Barton *et al.*, 1992). Phylogenetic methods which control for intra-specific variation by incorporating the uncertainty in to the error term are now available (Ives, Midford and Garland, 2007). Future work could exploit this development, if and when sufficient reliable data for sampling intraspecific variance become available for a large sample of species. However, this would in one sense only make the problem we have highlighted worse: the inflation of error terms that inevitably result can be expected to reduce the likelihood of finding significant correlations. The point we wish to emphasise here, however, is that current inferences in the literature about the selection pressures driving the evolution of brain size made using the standard approach of analysing single datasets appear to be unreliable. This point has important implications both for interpreting the existing literature, and for the design of future studies. Where variables are prone to measurement error and/or extensive intraspecific variation, such as is particularly likely to be the case with many behavioural variables, we recommend careful attention to data quality, testing the stability of results across datasets and/or incorporation of uncertainty in estimation of species-typical mean values.

In addition, statistical power is a serious issue where a range of predictors are considered with moderate or small numbers of species, as is not uncommonly the case in published comparative studies. In this situation (model overfitting) we can expect models with high coefficients of determination but poor generalizability from one dataset to another. This is a particular issue with the relatively small dataset of Stephan et al. (Stephan, Frahm and Baron, 1981), which has been the main empirical foundation for the claim that social group size is the strongest predictor of brain and/or neocortex size (Dunbar, 1992; Kudo and Dunbar, 2001; Dunbar and Shultz, 2007a; Dunbar and Shultz, 2017). When datasets 1 and 2 were matched to the species in the Stephan et al. data, the best models identified by our model comparisons did include group size (Appendices 10 & 11), in contrast with our results for the larger datasets. Hence, in accord with the suggestion of Parker that this dataset may be biased in favour of the SBH (Parker, 2015), we recover a clear correlation with group size only when analysis is restricted to these species. It therefore seems that the differences in patterns of correlations between studies (Dunbar and Shultz, 2007a; DeCasien *et al.*, 2017) are at least partly due to different species sampling and/or different predictor variables, rather than simply to use of different brain measures (overall brain size versus neocortex size).

3.4.4 Difficulties of a singular explanation of brain size variation

The fact that an effect of home range size emerges through two different types of analysis and two different (albeit not independent) datasets may make it tempting to interpret ranging as the "true" correlate of primate brain size, and to suggest, as others have done, that large brains reflect selection on spatial memory (Shultz and Dunbar, 2006; Dunbar and Shultz, 2007b). We, however, urge caution in this respect. First, we cannot unambiguously separate the effects of home range size, diet and activity period. Second, and in our view more importantly, overall brain size does not necessarily reflect the ways in which different selection pressures acted on different neural systems (Barton, Purvis and Harvey, 1995; Barton and Harvey, 2000; Healy and Rowe, 2007). For example, we found evidence that diurnality is associated with larger brains, but this result was weak and lacking consistency across datasets. Evolutionary transitions between nocturnal and diurnal niches are known to correlate with the relative size of visual and olfactory brain regions (Barton, Purvis and Harvey, 1995). Crucially, visual and olfactory regions show opposite evolutionary patterns (the former being relatively large and the latter relatively small in diurnal species), so that overall brain size fails to adequately capture the influence of sensory niche on information-processing capacities (Barton, Purvis and Harvey, 1995). In this case, the relatively weak and

variable effects of activity period on overall brain size can only be interpreted by understanding the divergent responses of underlying neural systems. Similarly, recent evidence reveals a striking difference in the pattern of brain component evolution in apes compared to other anthropoid primates, with increased cerebellar relative to cortical expansion in the former (Barton and Venditti, 2014). These different neural causes of brain size variation in different clades can be presumed to have different cognitive implications, presenting a difficulty for the attempt to relate overall brain size to individual selection pressures (Healy and Rowe, 2007) or to some general cognitive ability. While large brain regions such as the mammalian neocortex and avian pallium inevitably have a relatively strong impact on overall brain size (Sayol, Lefebvre and Sol, 2016), these components themselves consist of multiple functional systems that evolve in a mosaic fashion in response to different selection pressures (Barton, Purvis and Harvey, 1995; Barton, 2007; Montgomery, Mundy and Barton, 2016; Sayol, Lefebvre and Sol, 2016; Carlisle *et al.*, 2017; Logan *et al.*, 2017; Moore and DeVoogd, 2017). Making sense of the behavioural and ecological correlates of brain size will therefore depend on the difficult task of understanding the complex and clade-specific ways in which brain size reflects variation in specific neural systems.

4 The behavioural ecology of primate brain structures

4.1 Introduction

4.1.1 Mosaic change and the problem of linking function to whole brain size

Much of the early comparative work on the selection pressures that shape brains focused on whole brain size (Eisenberg and Wilson, 1978; Clutton-Brock and Harvey, 1980; Harvey, Clutton-Brock and Mace, 1980; Martin, 1984). This measure is still the metric of choice in many recent studies examining neural evolution in relation to ecological and behavioural specialisations (Shultz and Dunbar, 2006; Pérez-Barbería, Shultz and Dunbar, 2007; Dunbar and Shultz, 2007a; Kotrschal *et al.*, 2013, 2015; Benson-Amram *et al.*, 2016; Heldstab *et al.*, 2016; Sayol, Lefebvre and Sol, 2016; DeCasien *et al.*, 2017). Using a cognitive measure derived from a meta-analysis of the literature on "intelligence" and "cognitive ability", Deaner et al. demonstrated that "general cognitive ability", a composite measure derived from multiple cognitive tests, correlated best with measures of whole brain size rather than measures of individual structures like the neocortex (Deaner *et al.*, 2007). However, brain size has been criticised as a measure, as it potentially masks informative variation within the brain, and it is unclear by what mechanism changes in brain size translate in to changes in behaviour (Healy and Rowe, 2007; Logan *et al.*, 2017). A number of studies have found that volumes of individual structures are more closely linked to function than is overall brain size (Dechmann and Safi, 2009; Swanson *et al.*, 2012; Logan *et al.*, 2017). Change in particular structures may not always be positively correlated and therefore not necessarily reflected in overall brain size measures. Barton et al. (Barton, Purvis and Harvey, 1995) found such a pattern in the olfactory and visual systems of primates; enlargement in visual systems is matched by a reduction in olfactory structure sizes. Inappropriately treating the brain as a functionally and anatomically homogeneous organ may therefore frustrate studies which seek to examine functional brain variation. Rather, the brain is composed of a number of anatomically distinct structures and evidence suggests these have undergone change independently of one another. Examining variation in brain structures may therefore reveal more robust correlations.

4.1.2 Mosaic brain evolution: structure level and system level

Around 10% of size change at the level of major structures is independent of size change in the rest of the brain (Barton, 2009). Hence, "mosaic evolution" (Barton and Harvey, 2000; de Winter and Oxnard, 2001; Kolb *et al.*, 2013; Herculano-Houzel, Manger and Kaas, 2014; Moore and DeVoogd, 2017) of these major brain structures is thought to take place "in the context of otherwise concerted scaling" (Herculano-Houzel, Manger and Kaas, 2014). Selection on behaviour can cause change in the specific structures which underpin its function (Jerison, 1973), causing mosaic change independent of other structures and of the rest of the brain. This pattern is perhaps most strikingly manifest in distantly related taxa which have converged in aspects of their brain structure due to sharing a common lifestyle. One such example is demonstrated by de Winter and Oxnard (de Winter and Oxnard, 2001), who found that a number of Old and New World bat species that had independently become nectivorous had also converged on similar brain structure proportions. Evidence of mosaic brain evolution has also been shown in taxa with divergent lifestyles and divergent brain composition: fossorial mammals have reduced visual structures, while aquatic mammals have undergone a reduction in olfactory structure sizes (Barton, Purvis and Harvey, 1995). An interesting experimental example comes from a study on mice which were selectively bred for a high rate of wheel running. The midbrains of these mice were larger relative to the rest of the brain than the non-selectively bred control group, demonstrating that selection for a behaviour had influenced the volume of a specific brain area (Kolb *et al.*, 2013).

This mosaic pattern can operate at two levels: at the level of individual structures and of neural systems formed of groups of anatomically and functionally linked structures (Montgomery, Mundy and Barton, 2016). Firstly, individual structures can change independently of the rest of the brain. This is apparent in the differences in brain composition between clades (de Winter and Oxnard, 2001; Herculano-Houzel, Manger and Kaas, 2014). Barton and Harvey demonstrated these grade shifts in relative (to the rest of the brain) neocortex size between insectivores, strepsirrhine primates and haplorhine primates, indicating that the relationship between the neocortex and the rest of the brain varies both within and between orders (Barton and Harvey, 2000). Similarly, the relative size of the cerebellum is larger in apes than in non-apes (Rilling and Insel, 1998; Barton and Venditti, 2014), and despite having near equal brain sizes, the tectum of the squirrel is around 10 times larger than that of a rat (Kaas and Collins, 2001).

While these examples show that structures do to some extent evolve independently, they do not function entirely in isolation. These are densely interconnected and functionally specialised systems that are distributed across multiple areas (Buckner and Krienen, 2013). Such functionally linked systems are the second level at which mosaic brain evolution operates. Functionally and anatomically linked structures can coevolve independently of change elsewhere in the brain (Harvey and Krebs, 1990; Barton and Harvey, 2000; Barton, 2009) in response to selective pressures and constraints. This is most notably observed in the primate cortico-cerebellar system. The neocortex has long been at the centre of the discussion of primate brain evolution due to its large size relative to the rest of the brain (Dunbar, 1992). The cerebellum has been relatively neglected and had long been thought of as simply a "motor structure", responsible for the management of motor learning and balance (Sultan and Glickstein, 2007; Barton, 2012). However, increasingly sophisticated imaging techniques have shown that the cerebellum has connections not only with the primary motor cortex but also to prefrontal, superior temporal and posterior parietal lobes (Ramnani, 2006; Cantalupo and Hopkins, 2010); cortical areas with a breadth of functionality. The increasingly apparent role of the cerebellum in diverse types of cognition (MacLeod *et al.*, 2003; Whiting and Barton, 2003; Ramnani, 2006; Cantalupo and Hopkins, 2010; Herculano-Houzel, 2010; Barton, 2012; Hall, Street and Healy, 2013; Koziol *et al.*, 2013; Stoodley and Schmahmann, 2016) should therefore be understood through its relationship with the neocortex. Anatomically distinct loops exist between the two structures (Ramnani, 2006), indicating integrated information processing across the two structures. The clearest indication of the neocortex, cerebellum and intermediate nuclei forming a functional system is the correlated evolution of the two structures (Whiting and Barton, 2003; Herculano-Houzel, 2010), changing size in concert with each other in part independently of other structures (but see Barton & Venditti (2014) for a different pattern in apes). The shared functional capacity between these two structures suggests that their shared volumetric variation may correlate independently with behavioural ecological variables. This is explored in the coming analyses.

4.1.3 Hypotheses for the evolution of whole brain size: implications for specific structures

4.1.3.1 Ecological hypotheses

The hypotheses relating to variation in whole brain size explored in Chapter 2 also have implications for specific neural structures. Ecological hypotheses emphasise the role of the ecological niche in brain size variation. As discussed in Chapter 2, diet is arguably the

variable most central to ecological hypotheses of brain evolution. Its effect on brain size was identified early in the literature by Clutton-Brock and Harvey (1980) and has again been implicated in more recent work using up-to-date Bayesian phylogenetic analysis and much larger sample sizes (DeCasien *et al.*, 2017). There is some disagreement as to whether diet is a pressure or a constraint on brain size; for example it has been suggested that frugivores have larger brains than folivores because fruit is less predictably distributed and so imposes a greater cognitive load requiring larger brains (Harvey, Clutton-Brock and Mace, 1980; Milton, 1988), but it has also been suggested that diet constrains brain size because neural tissue is energetically expensive and different diets have different energy densities (Aiello and Wheeler, 1995). Home range size also features in ecological hypotheses and has direct links to diet, as species whose preferred food type is widely dispersed require a larger home range to acquire sufficient energy (Milton and May, 1976; Milton, 1988). Home range size correlates with relative brain volume (Clutton-Brock and Harvey, 1980), in support of spatial/foraging hypotheses which posit that large brains are linked to the spatial cognition and memory demands associated with navigating a large home range (Milton and May, 1976; Clutton-Brock and Harvey, 1980; Milton, 1988; Parker, 2015). Ecological hypotheses are often associated with whole brain size rather than explicitly linked to specific structures (Harvey and Krebs, 1990; Dunbar, 1992), but spatial/foraging hypotheses have implicated the hippocampus in particular (Harvey and Krebs, 1990; Hopkins, Lyn and Cantalupo, 2009) as its size has been shown to correlate with spatial cognition and memory (Healy and Krebs, 1992; Sherry, Jacobs and Gaulin, 1992; Clayton, Reboreda and Kacelnik, 1997; Parker, 2015). The neocortex and cerebellum are also involved in visuo-spatial processing (MacLeod *et al.*, 2003; Glickstein, Sultan and Voogd, 2011; Koziol *et al.*, 2013) and so may be predicted to relate to ranging in terms of orientation in and movement through space.

4.1.3.2 The Social Brain Hypothesis

Perhaps the most well-known relationship between a brain structure and behaviour is the oft-cited correlation between neocortex and group size that formed the basis of the Social Brain Hypothesis (SBH) (Dunbar, 1992; Dunbar, 1998; Dunbar and Shultz, 2007b). As described in previous chapters, the SBH suggests that managing social relationships requires complex computation and thus larger brains. Group size has been shown to correlate with both whole brain (Shultz and Dunbar, 2006, 2010; Pérez-Barbería, Shultz and Dunbar, 2007) and neocortex size (Barton and Dunbar, 1997; Shultz and Dunbar, 2006; Dunbar and Shultz, 2007a). In contrast to the early incarnation of the SBH by Brothers (1990), Dunbar (mostly)

focused specifically on the neocortex, suggesting it was "the 'thinking' part of the brain" and so its volume was an appropriate "index of cognitive ability" (Dunbar, 1992, p. 473). He suggested that the cognitive demands of managing complex and numerous relationships requires a large brain or neocortex (Dunbar and Shultz, 2007a). He found that neocortex volume and group size were highly correlated (Dunbar, 1992; Dunbar and Shultz, 2007b); a finding which has been both supported (Barton, 1996; Kudo and Dunbar, 2001; Reader and Laland, 2002; Byrne and Corp, 2004; Walker *et al.*, 2006; Sallet *et al.*, 2011; Powell *et al.*, 2012; Arsznov and Sakai, 2013) and disputed (Clutton-Brock and Harvey, 1980; Swanson *et al.*, 2012; Holekamp *et al.*, 2015) since.

4.1.3.3 The Visual Brain

The "Visual Brain Hypothesis" (Barton, 1998) emphasises the role of primates' visual specialisation; suggesting that a large proportion of the variance in primate brain size can be attributed to visual adaptations. For example, 50% of the neocortex of macaques is comprised of visual areas (Barton, 1996, 1998). The cortex receives visual information from the lateral geniculate nucleus (LGN) of the thalamus which, along with the visual cortex, is disproportionately enlarged in large-brained primates, and in diurnal primate taxa (Barton, Purvis and Harvey, 1995; Barton, 2006b).

4.1.3.3.1 The eco-visual brain

The enlargement of the primate neocortex is specifically associated with the expansion of the parvocellular pathway of the LGN, which is principally associated with high acuity photic vision and colour discrimination (Barton, 1998). The volume of the parvocellular layers of the LGN correlate with frugivory (Barton, 1998). This, coupled with the known positive association between frugivory and relative brain size (Clutton-Brock and Harvey, 1980), has led to suggestions that this visual specialisation is in part an adaptation in diurnal frugivorous primates for discerning appropriate food sources (Barton, 1998). This sub-hypothesis of the Visual Brain which interprets visual specialisation as adaptive for ecological reasons in hereafter referred to as the "eco-visual brain".

4.1.3.3.2 The socio-visual brain

The parvocellular LGN volume also correlates with activity period (diurnal species have larger LGNs) and group size (species with larger LGNs tend to live in larger groups). Diurnal taxa tend to have larger group sizes (Barton, 1996). Barton has suggested that the specific association of the parvocellular layers of the LGN (which project to the visual areas of the

cortex) and group size might explain the correlation between neocortex size and group size which formed the basis of Dunbar's Social Brain Hypothesis (Dunbar, 1992; Dunbar, 1998; Dunbar and Shultz, 2007b). He suggests that processing socio-visual cues such as facial expression and body language requires the fine discrimination afforded by the parvocellular pathway (Barton, 1998, 2009). In support of a role for sociality in the evolution of the visual brain, Dobson and Sherwood (2011) reported evidence of correlated evolution between facial motor control, group size and the volume of area V1 (the primary visual cortex) in Catarrhini. More recently, Hiramatsu and colleagues have reported that trichromacy facilitates the recognition of primate facial signals (Hiramatsu *et al.*, 2017). This interpretation of the Visual Brain is hereafter referred to as the "socio-visual brain".

4.1.4 Predictions for associations between structures and behavioural-ecological variables

The hypotheses giving rise to these predictions are given in parentheses.

Group size

1. Neocortex volume is predicted to correlate positively with group size (Social Brain)
2. LGN volume is predicted to correlate positively with group size (Socio-visual brain).

Diet

3. LGN is predicted to correlate negatively with folivory (Eco-visual brain).

Home range size

4. Hippocampus volume is predicted to correlate positively with home range size (Ecological - spatial memory).
5. Neocortex volume is predicted to correlate positively with home range size (Ecological).
6. Cerebellum volume is predicted to correlate positively with home range size (Ecological).

Activity period

7. Neocortex and LGN volume predicted to correlate positively with diurnality (Eco-visual brain).

Since the thalamus and striatum both form part of feedback loops with the neocortex, they were predicted to follow the same patterns of behavioural-ecological correlations. Despite

not having independent predictions, it was reasoned that they were structures of significant importance to the systems of interest (most notably the cortico-cerebellar system) and so warranted inclusion in the analyses.

4.1.5 This study

This chapter will refine the analyses of the previous chapter by examining changes in the brain at finer scale using volumetric data on individual structures rather than whole brain measures. The analyses will again explore the relationships between behavioural-ecological data drawn from two sources and structure size, but this time using a new, more up-to-date volumetric dataset gathered with modern methods. A composite variable combining the neocortex and cerebellum will also be included to assess whether this functional complex is more sensitive to changes in behaviour and ecology than the individual structures. Analyses are performed firstly with body mass (g) as a covariate to control for overall size. This analysis allows examination of whether structures vary similarly with behavioural correlates, allowing for the possibility of functionally linked complexes of structures to be accounted for. The second phase of analysis includes a measure of the volume of the rest of the brain (the volume of the brain excluding the structure of interest) as well as body mass, which allows examination of how each structure varies in relation to remaining brain size. For clarity in the later discussion, only results pertinent to the hypotheses examined above are discussed.

4.2 Methodology

4.2.1 Volumetric brain structure data

Volumetric data for primate brain structures were obtained from a new dataset collected by Navarette (pers. comm.). This dataset was collected to update and augment an existing widely used comparative dataset published by Stephan, Frahm and Baron (1981), using post mortem MRI scans to take volumetric measurements. The Navarette data were combined with the Stephan data to form one large updated dataset. Where figures were available in both datasets, a mean was taken. The structures included were neocortex, cerebellum, hippocampus, thalamus, striatum, lateral geniculate nucleus (LGN) and a composite variable "cortex + cerebellum" which is the sum of neocortex and cerebellum volume. This cortex + cerebellum measure is included as these two structures exhibit particularly strongly correlated

volumetric evolution (Whiting and Barton, 2003). Although sample size and missing data precluded the inclusion of other structures involved in this system such as the thalamus, pons and vestibular nuclei, this measure included the two largest structures in the system and so is likely to show the same correlations as the whole complex (Whiting and Barton, 2003). The structures chosen for analysis were based on their relevance to major brain evolution hypotheses, whilst balancing data availability and sample size. The selected structures had good sample sizes (62 species) and representation of important functional areas. Paired t-tests on species that appeared in both datasets revealed no significant difference between the Navarette data and the Stephan data except in the case of the medulla and pons (Appendix 21). These structures were therefore excluded from the analyses

4.2.2 .Behavioural-ecological data

As in Chapter 3 we used two behavioural-ecological datasets in order to assess stability of results. The first, collected by Isler (Powell, Isler and Barton, 2017) will hereafter be referred to as dataset 1. The second, collected by Nunn and van Schaik (2002) will be referred to as dataset 2. Data on four behavioural ecological variables and body size were drawn from these two datasets. A combined dataset was also constructed, taking the mean of values reported in datasets 1 and 2 and filling gaps in the data with figures from additional literature in order to create a larger, more representative dataset. This combined dataset (hereafter referred to as dataset 3) represents the largest sample (n=62). Datasets 1 and 2 are analysed for the purposes of comparing their results to assess the robustness of results to alternative datasets, whereas dataset 3 serves as the larger and more comprehensive dataset and so is used to give more definitive results.

The behavioural-ecological variables selected for analysis were diet (folivory vs non folivory), activity period (diurnality vs nocturnality), group size and home range size (ha). Diet is highly variable, changing within species from one population to another, and also within individuals with seasonal availability. It was therefore reasoned that the most biologically grounded way to categorise diet was by folivores versus non folivores, as folivory is a dietary strategy which requires specialist gut and dental adaptations (Hladik, 1978; Chivers and Hladik, 1980; Fleagle, 2013) and so is invariant between and within individuals. In datasets 1 and 2, diet was categorised by the food type that comprised the largest proportion of a species' intake. In dataset 3, folivores were more strictly defined as only those species with anatomical and physiological adaptations to processing non-reproductive plant matter, such as complex sacculated stomachs and enlarged colons (Chivers

and Hladik, 1980), and well developed molar shearing crests (Fleagle, 2013). This was done to avoid possible issues associated with dietary categorisation based on behavioural data, such as biases from inadequate assessment of seasonal fluctuations and other sources of intraspecific variation.

Activity period was treated as a categorical variable with two levels: diurnality and nocturnality. As in the previous chapter, cathemeral species are incorporated in the diurnal category. The diurnal category therefore encompassed all taxa which exhibit any substantive diurnal behaviour. Home range size and group size are treated as continuous variables. As discussed in the "Ecological Hypotheses" section above, home range size has a relationship with diet as the distribution of resources has a direct impact on the necessary size of an animal's range. Therefore, including home range size in the current analyses is required both to analyse its independent effect on the volumes of structures but also to control for its effect on other behavioural ecological variables.

Statistical power is a major issue for comparative studies where sample sizes are often small. This, coupled with the need to include large numbers of parameters (either for predictive or control purposes) can make results difficult to interpret (Borries *et al.*, 2016). As terrestriality did not show any significant relationships with overall brain size or feature in any of the models selected by model comparison in the previous chapter, it was omitted to reduce the number of parameters.

4.2.3 Statistical analysis

Allometric effects were controlled for by including body size (g) as a covariate in the first set of regressions. If a brain structure is part of a distributed system, the other structures in the system may undergo the same size change as the structure of interest. If brain size is then controlled for, the structure may appear not to have changed size relative to the rest of the brain. Thus, including a set of analyses which control for size by only using body size allows for the possibility of associations between functionally linked structures and behavioural ecological variables – mosaic brain evolution at the level of the system. This condition is hereafter referred to as the "body size corrected" condition. A second set of analyses including the "rest of brain" volume (i.e. the volume of the structure of interest subtracted from the total brain volume) as covariate was then also included to examine whether structures vary independently of the rest of the brain – mosaic brain evolution at the level of

the structure. This condition is hereafter referred to as the "RoB corrected" condition; meaning rest of brain (RoB) is included as a covariate along with body mass.

As mentioned in the introductory chapter, when controlling for size some comparative neuroanatomists have preferred structures which are thought to be relatively conserved across evolutionary time such as the brain stem (Dunbar, 1992; Reader and Laland, 2002) and spinal cord (Willemet, 2013). Using these structures has the advantage of being able to use the same control structure for every test, rather the RoB which necessarily changes each time. However, few species have complete data for these structures, so their inclusion would dramatically reduce sample size. Phylogenetic least squares (PGLS) regression was employed to analyse the relationships between behavioural-ecological variables and brain structure volumes while controlling for the confounding effects of phylogeny. The phylogeny used was the consensus tree from 10k Trees (Arnold, Matthews and Nunn, 2010). Data were analysed in R (R Development Core Team, 2015) using the 'ape' (Paradis, Claude and Strimmer, 2004), 'caper' (Orme *et al.*, 2013), 'geiger' (Harmon *et al.*, 2008), 'nlme' (Pinheiro *et al.*, 2015) and 'lmtest' (Zeileis and Hothorn, 2002) packages. Regression models were subjected to model comparisons using AIC (Aikaike's Information Criterion) values and log likelihood ratio tests. Only dataset 3 was subjected to model comparisons as it had the largest sample and was a composite of dataset 1 and 2 which were analysed separately for comparative purposes. AIC was utilised as it penalises overparameterised models. This comparison indicates whether more complex models are warranted by assigning each model a criterion value by which each model's fit can be compared to the rest. Lower AIC values indicate a better model in terms of fit and parsimony (Burnham and Anderson, 2002). Log likelihood ratio tests were also used, as these allow comparison of each model to a null hypothesis, therefore giving an absolute measure of fit. This is in contrast with the AIC which provides a measure of fit which is relative to the other tested models, meaning if all models constitute a poor fit, AIC alone will not reveal this (Maydeu-Olivares and García-Forero, 2010). Models ranging from least to most complex (in terms of numbers of variables) were compared, with variables entered in to log likelihood ratio tests in order of their p value (smallest to largest) derived from simple models including each variable alone with body size.

Variance Inflation Factors (VIFs) were calculated using the R package 'car' (Fox and Weisberg, 2011) to check for cases of high multicollinearity. Multicollinearity can render results difficult to interpret as highly collinear variables share a lot of variance, making it difficult to assess their relative importance and reducing statistical power (Field, Miles and

Field, 2012). There is currently no package for calculating VIFs phylogenetically, so these were performed without correcting for the non-independence of data points. This is actually a more conservative test for collinearity as correlations would be larger without accounting for phylogeny (Freckleton, Harvey and Pagel, 2002), increasing VIF values. Including a rest of brain measure in an analysis together with body size is problematic as the two are highly correlated. This was confirmed in the datasets used in this study by very large VIFs (>16). However, as these were covariates rather than predictors, it seems likely that their collinearity did not affect interpretation of the effects of the behavioural ecological correlates. Size was controlled for, but the collinearity of the two size measures meant we could not interpret their independent correlations with structure volumes. Excluding the covariates, VIFS were less than 4.1 in all cases. The largest correlation was between home range size and body size in dataset 3 at .75. However, collinear predictors with absolute correlations under .8 are not thought to be problematic in PGLS (Garland, 2012). Although the correlation was quite large, the VIF of 4.1 was small enough to warrant keeping this predictor in the analyses (Quinn and Keough, 2002).

4.3 Results

4.3.1 Home range size and diet

The body size corrected analyses of the neocortex, cerebellum, and cortex + cerebellum show a pattern of substitution between home range size (HRS) and diet; with diet reaching significance in dataset 1 (the relationship with cerebellum volume narrowly misses significance in PGLS analyses but is supported by model comparisons in Appendix 25) and HRS reaching significance in dataset 2. Home range size was the only predictor which significantly improved the fit of models of cerebellum and neocortex + cerebellum relative to allometric models (Appendices 25 & 26).

In dataset 3, model comparisons for the neocortex showed that diet was an informative predictor, but HRS just missed significance (p=0.06, Appendix 22). Home range size and diet therefore seem to have a relationship which results in this switching pattern (although as the bivariate correlations (Appendices 36, 37 & 38) reveal no correlation between these two variables, this relationship may be indirect). This mirrors the results of the previous chapter where a similar pattern of mutual substitution was observed in the relationships of diet and home range size with whole brain size.

4.3.2 Home range size

In the RoB corrected analyses HRS was no longer a significant predictor of any structure's volume and did not form part of any of the "best" models in model comparisons, except in the case of the striatum in dataset 3 (Table 4.3-6 & Appendix 31). The pattern of substitution between diet and home range size was not evident in the RoB corrected analyses (Tables 4.3-4, 4.3-5 & 4.3-6).

4.3.3 Activity period

Activity period overall showed a fairly consistent association with lateral geniculate nucleus and neocortex volume, as predicted. In the body size controlled condition, the lateral geniculate nucleus was positively associated with activity period (diurnality) in dataset 1 as predicted but did not reach significance in datasets 2 and 3 (Tables 4.3-1 – 4.3-3. However, model comparisons on dataset 3 show activity period does improve the fit of a model of LGN volume over body size alone (Appendix 28).

Despite not always reaching significance in PGLS, neocortex and activity period appear to show a fairly consistent relationship, with diurnal species tending to have larger neocortices. In dataset 3, the log likelihood ratio tests showed that both activity period and diet significantly improved fit of a model of neocortex volume in both the body size corrected and brain size corrected analyses. However, neither reached significance in the PGLS analyses of dataset 3 (Tables 4.3-3 & 4.3-6). Activity period was a significant predictor when body size was controlled for in dataset 2, but not when RoB was controlled for. Diet was a significant predictor of neocortex volume across both covariate conditions in dataset 1.

4.3.4 Group size

The most consistent result for group size was the positive relationship with thalamus volume, which was present across all three datasets when the rest of brain volume was not controlled for (Tables 4.3-1 – 4.3-3). When the rest of brain volume was included, this relationship failed to reach significance in datasets 1 and 2 but was present in dataset 3, possibly due to the increased sample size and statistical power (dataset 3: n=62; dataset 1: n=52; dataset 2: n=47). The model comparisons for both body size corrected and RoB corrected conditions showed that group size was the only predictor whose inclusion improved the fit of the thalamus volume model relative to a purely allometric model (Appendices 27 & 34).

The striatum showed a similar relationship with group size but was not quite as consistent, only reaching significance in dataset 1 but also narrowly missing it in dataset 3. Model comparisons based on log likelihood ratios showed the same pattern as the thalamus; group size was the only informative predictor (Appendix 24). The PGLS results did not show any associations between group size and neocortex size in either size correction condition.

Table 4.3-1 - Body size corrected PGLS regressions of brain structure volumes on behavioural-ecological variables in dataset 1 (n=52)

	Neocortex		Cerebellum		Cortex + cerebellum		Hippocampus		Striatum		Thalamus		LGN	
Predictor	t_{46}	*p*	t_{46}	*p*	t_{46}	*p*	t_{46}	*p*	t_{46}	*p*	t_{39}	*p*	t_{43}	*p*
Intercept	7.13	**<0.001‡**	7.75	**<0.001‡**	10	**<0.001‡**	7.6	**<0.001‡**	4.44	**<0.001‡**	5.39	**<0.001‡**	0.43	**<0.001‡**
Body Size	10.57	**<0.001‡**	14.89	**<0.001‡**	13.58	**<0.001‡**	9.7	**<0.001‡**	12.17	**<0.001‡**	9.94	**<0.001‡**	9.45	**<0.001‡**
Diurnality	1.9	0.06	0.79	0.44	1.74	0.09	-2.14	**<0.05***	0.6	0.55	0.67	0.5	2.51	**<0.05***
Folivory	-2.69	**<0.01†**	-1.99	0.05	-2.65	**<0.05***	0.43	0.67	-1.61	0.11	-1.08	0.29	-1.13	0.26
Group Size	0.8	0.43	1.13	0.26	1.13	0.27	-1.44	0.16	2.83	**<0.01†**	2.62	**<0.05***	1.86	0.06
HRS	-0.07	0.95	0.48	0.63	-0.26	0.79	0.75	0.46	-1.31	0.2	-0.16	0.88	-0.12	0.91
Model Summary														
λ	.36		.91		.58		.00		.92		.94		.73	
r^2	.83		.89		.88		.83		.82		.80		.80	

Bold denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
***** = p <0.05, **†** = p <0.01. **‡** = p <0.001

Table 4.3-2 - Body size corrected PGLS regressions of brain structure volumes on behavioural-ecological variables in dataset 2 (n=47)

	Neocortex		Cerebellum		Cortex + cerebellum		Hippocampus		Striatum		Thalamus		LGN	
Predictor	t_{41}	*p*	t_{41}	*p*	t_{41}	*p*	t_{41}	*p*	t_{41}	*p*	t_{34}	*p*	t_{38}	*p*
Intercept	14.73	**<0.001‡**	12.47	**<0.001‡**	15.2	**<0.001‡**	10.5	**<0.001‡**	10.31	**<0.001‡**	7.34	**<0.001‡**	3.06	**<0.001‡**
Body Size	1.58	**<0.001‡**	2.01	0.05	1.65	0.11	0.26	0.8	1.3	0.2	1.65	0.11	5.26	**<0.001‡**
Diurnality	2.14	**<0.05***	1.89	0.07	2.1	**<0.05***	1.01	0.32	1.75	0.09	2.89	**<0.01†**	0.58	0.57
Folivory	-0.1	0.92	-0.4	0.69	-1.15	0.88	0.57	0.58	-0.21	0.83	-0.47	0.64	-1.84	0.07
Group Size	0.15	0.88	-0.09	0.93	0.14	0.88	-0.29	0.77	0.74	0.47	2.12	**<0.05***	1.49	0.14
HRS	3.14	**<0.01†**	3.35	**<0.01†**	3.16	**<0.01†**	3.16	**0.01†**	2.51	**<0.05***	1.79	0.08	2.02	0.05
Model Summary														
λ	.96		1		.97		.90		.98		1		.88	
r^2	.35		.36		.36		.21		.27				.68	

Bold denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
***** = p <0.05, **†** = p <0.01. **‡** = p <0.001

Table 4.3-3 - Body size corrected PGLS regressions of brain structure volumes on behavioural ecological variables in dataset 3 (n=62)

	Neocortex		Cerebellum		Cortex + Cerebellum		Hippocampus		Striatum		Thalamus		LGN	
Predictor	t_{56}	*p*	t_{56}	*p*	t_{56}	*p*	t_{56}	*p*	t_{56}	*p*	t_{46}	*p*	t_{52}	*p*
Intercept	9.18	**<0.001‡**	9.24	**<0.001‡**	11.99	**<0.001‡**	7.68	**<0.001‡**	5.85	**<0.001‡**	6.12	**<0.001‡**	3.01	**<0.01†**
Body Size	9.43	**<0.001‡**	11.5	**<0.001‡**	11.32	**<0.001‡**	8.44	**<0.001‡**	10.8	**<0.001‡**	8.71	**<0.001‡**	5.27	**<0.001‡**
Diurnality	1.76	0.08	0.74	0.46	1.49	0.14	-1.84	0.07	0.54	0.59	0.74	0.46	1.94	0.06
Folivory	-1.8	0.08	-0.16	0.88	-1.36	0.18	1.44	0.17	-1.08	0.28	-0.08	0.93	-1.24	0.22
Group Size	0.55	0.61	0.13	0.75	0.74	0.46	-1.25	0.22	1.89	0.06	3.2	**<0.001‡**	0.66	0.51
HRS	1.3	0.2	2.28	**<0.05***	1.5	0.13	2	0.28	0.6	0.55	0.41	0.68	1.92	0.06
Model Summary														
λ	.38		91		.59		.00		.75		.95		.92	
r^2	.82		.82		.35		.81		.80		.73		.68	

Bold denotes significance at at least the α<0.05 level. Degrees of freedom are indicated in subscript after "*t*"
***** = p <0.05, **†** = p <0.01. **‡** = p <0.001

Table 4.3-4 - Body and rest of brain size corrected PGLS regressions of brain structure volumes on behavioural-ecological variables in dataset 1

	Neocortex		Cerebellum		Cortex + cerebellum		Hippocampus		Striatum		Thalamus		LGN	
Predictor	t_{45}	*p*	t_{45}	*p*	t_{45}	*p*	t_{45}	*p*	t_{45}	*p*	t_{38}	*p*	t_{42}	*p*
Intercept	3.85	**<0.001‡**	-2.73	**<0.01†**	7.09	**<0.05***	2.58	**<0.05***	-4.04	**<0.001‡**	-1.84	0.07	-4.74	**<0.001‡**
Body Size	5.09	**<0.001‡**	1.37	0.18	9.94	**<0.001‡**	3.07	**<0.01†**	0.01	0.99	0.07	0.95	0.68	0.5
RoB	-0.91	0.37	8.59	**<0.001‡**	-2.47	**<0.05***	-0.2	0.84	7.23	**<0.001‡**	5.04	**<0.001‡**	5.34	**<0.001‡**
Diurnality	1.93	0.06	-0.65	0.52	1.92	0.06	-2.03	**<0.05***	-0.82	0.42	-0.86	0.39	2.25	**<0.05***
Folivory	-2.66	**<0.05***	0.29	0.78	-2.21	**<0.05***	0.27	0.79	0.35	0.73	0.54	0.59	0.61	0.55
Group Size	0.84	0.4	-0.21	0.83	1.57	0.12	-1.28	0.21	1.65	0.11	1.66	0.1	1.37	0.18
HRS	0.004	1	1.36	0.18	-0.95	0.35	0.74	0.46	-0.99	0.33	0.57	0.57	-0.34	0.73
Model Summary														
λ	.47		.8		.97		.00		.7		.82		.75	
r2	.81		.96		.81		.82		.94		.9		.88	

Bold denotes significance at at least the α<0.05 level. ***** = p <0.05, **†** = p <0.01. **‡** = p <0.001

Degrees of freedom are indicated in subscript after "*t*". RoB = rest of brain (total brain volume minus response structure)

Table 4.3-5 - Body and rest of brain size corrected PGLS regressions of brain structure volumes on behavioural-ecological variables in dataset 2 including rest of brain

	Neocortex		Cerebellum		Cortex + Cerebellum		Hippocampus		Striatum		Thalamus		LGN	
Predictor	t_{40}	*p*	t_{40}	*p*	t_{40}	*p*	t_{40}	*p*	t_{40}	*p*	t_{33}	*p*	t_{37}	*p*
Intercept	0.88	0.38	-5.47	**<0.001‡**	2.33	**<0.05***	-1.33	0.19	-7.53	**<0.001‡**	-4.6	**<0.001‡**	-1.07	0.29
Body Size	-0.13	0.9	0.94	0.35	0.46	0.65	-1.36	0.18	-2.19	**<0.05***	-0.74	0.47	0.51	0.61
RoB	12.86	**<0.001‡**	24.93	**<0.001‡**	10.44	**0.001‡**	7.2	**<0.001‡**	21.68	**<0.001‡**	13.37	**<0.001‡**	2.75	**<0.01†**
Diurnality	1.63	0.11	-1.14	0.26	1.12	0.27	-1.06	0.3	-1.28	0.21	0.09	0.93	0.41	0.68
Folivory	1.01	0.32	0.34	0.74	1.29	0.2	1.29	0.2	0.41	0.68	0.68	0.5	-0.6	0.55
Group Size	0.84	0.4	-0.04	0.97	0.58	0.57	-0.42	0.68	1.19	0.24	1.53	0.13	1.34	0.19
HRS	0.75	0.46	-0.28	0.78	0.89	0.38	0.21	0.83	-1.54	0.13	-0.98	0.33	1.35	0.18
Model Summary														
λ	.00		.85		.00		.51		.49		.76		.91	
r^2	.95		.96		.93		.68		.95		.92		.72	

Bold denotes significance at at least the α<0.05 level. ***** = p <0.05, **†** = p <0.01. **‡** = p <0.001
Degrees of freedom are indicated in subscript after "*t*". RoB = rest of brain (total brain volume minus response structure)

Table 4.3-6 - Body and rest of brain size corrected PGLS regressions of brain structure volumes on behavioural-ecological variables in dataset 3 including rest of brain

	Neocortex		**Cerebellum**		**Cortex + Cerebellum**		**Hippocampus**		**Striatum**		**Thalamus**		**LGN**	
Predictor	t_{55}	p	t_{55}	p	t_{55}	p	t_{55}	p	t_{55}	p	t_{45}	p	t_{51}	p
Intercept	3.44	**<0.01†**	-3.74	**<0.001‡**	5.85	**<0.001‡**	0.56	0.57	-5.64	**<0.001‡**	-2.54	**<0.05***	-2.15	**<0.05***
Body Size	3.96	**<0.001‡**	1.94	0.06	6.2	**<0.001‡**	1.29	0.2	0.29	0.77	1.81	0.08	0.56	0.58
RoB	1.09	0.28	12.88	**<0.001‡**	1.15	0.25	2.68	**<0.01†**	11.05	**<0.001‡**	7.15	**<0.001‡**	4.11	**<0.001‡**
Diurnality	1.75	0.08	-0.72	0.47	1.44	0.15	-2.46	**<0.05***	-0.53	0.6	-0.24	0.81	1.44	0.16
Folivory	-1.68	0.1	1.23	0.22	-1.22	0.23	2.26	**<0.05***	-0.2	0.84	0.08	0.94	-1.33	0.19
Group Size	0.58	0.56	-0.76	0.45	0.8	0.43	-1.84	0.07	3.02	**<0.01†**	3.03	**<0.01†**	0.53	0.6
HRS	0.99	0.33	0.08	0.94	1.19	0.24	0.58	0.56	-2.43	**<0.05***	-1.61	0.11	0.28	0.78
Model Summary														
λ	.28		85		.50		.00		.80		.92		.97	
r^2	.84		.96		.86		.83		.93		.88		.65	

Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01. ‡ = p <0.001
Degrees of freedom are indicated in subscript after "*t*". RoB = rest of brain (total brain volume minus response structure)

4.4 Discussion

I have examined the behavioural-ecological correlates of brain structure volumes. I used two comparative datasets of behavioural-ecological data and have used new, updated neuro-volumetric data. As in the previous chapter, there was variation in results across alternative datasets, likely due to issues of overparameterisation and data quality. Given these inconsistencies, any relationships discovered must be treated with a note of caution. However, despite this there were some results that showed some stability across or within datasets, and emergent patterns which are important for directing future work in this area.

4.4.1 Unexpected group size associations

4.4.1.1 Thalamus

The most consistent result across data sets was an unpredicted one; a relationship between group size and thalamus volume. This is the only relationship that shows good consistency across all three datasets, and both with and without the rest of brain measure, suggesting that this association is both robust and is independent of variation in the size of other brain regions. The relationship is also demonstrated in the model comparisons on dataset 3 which show that a model predicting thalamus volume from only body size and group size best describes the data, and that adding any further variables to the model did not improve the fit (Appendices 27 & 34). One might expect that the thalamus had a relationship with group size through its role in the corticocerebellar complex, but this is not the case since the association persists when the volume of the rest of the brain is controlled for (Table 4.3-6). There is some evidence in the comparative literature of a thalamic role in sociality; Armstrong, Clarke and Hill's 1974 study (Armstrong, Clarke and Hill, 1987) found that volumes of the anterior thalamic nucleus were larger in species that lived in single male groups than those in multi male groups, although the direction of this relationship is not consistent with the present results as single male groups are usually smaller than multi-male groups. The neuropsychological literature offers some more direct evidence of a link. For example, thalamic activation has been observed in response to social rejection as well as to physical pain (Hsu *et al.*, 2013). This study also suggested the striatum, along with the thalamus, formed parts of a system which modulated mood and motivation in response to social cues.

4.4.1.2 Striatum

In the current study, in the body size corrected condition, the striatum is also correlated with group size in dataset 1 (Table 4.3-1), and while it just fails to reach significance in dataset 3

(Table 4.3-3, p=0.06) the model comparisons show that group size alone contributes significantly to the fit in the striatum model in dataset 3 (Appendix 24). A similar picture emerges from the RoB controlled condition, where striatum again correlates with group size in the combined dataset 3 (Table 4.3-6). Abnormalities in the striatum and the thalamus are associated with generalised social anxiety disorder (van der Wee *et al.*, 2008). The model comparisons in the present study (Appendices 24, 27, 31 & 34) suggest that the volume of these structures may be linked to group size. These results seem to indicate that the relationship between the thalamus and the striatum with group size is reliable, although the striatum association is less robust. However, since these relationships were unpredicted these are post-hoc explanations further investigation is needed to establish whether and how these structures are related to sociality. A simple way to test this would be a PGLS regression with thalamus or striatum volume as the response variables and group size and rest of brain volume as predictors.

4.4.2 Group size: part of an adaptive syndrome?

There is some evidence of a substitution of group size and home range size effects on striatum volume across datasets. In datasets 1 and 2, the two variables switch places as the significant variable (when rest of brain is not controlled for). In dataset 3 when rest of brain is controlled for, there is a significant effect of both group size and home range size in the PGLS analysis. However, the log likelihood tests show that the inclusion of group size alone does not improve the fit over a purely allometric model (including only body size and RoB). The inclusion of home range size along with group size constitutes a significantly better fit (Appendix 31). The AIC model comparisons also show that the group size and home range size only models are not significantly different to each other. Other studies have also reported difficulty in separating the effect of home range size and group size on (whole) brain size (Deaner, Nunn and van Schaik, 2000; Walker *et al.*, 2006). The difficulty in separating effects of these predictors may be in part due to the fact that group size, home range size and diet are interlinked. Group size and home range size are correlated (as indicated in the bivariate correlations in Appendices 36, 37 and 38): larger groups tend to have larger home ranges, likely in order to have access to sufficient food sources to meet the group's energy demands (McNab, 1963; Clutton-Brock and Harvey, 1977; Nunn and van Schaik, 2002).

The pattern of home range size associations and group size "flipping" in this manner implies that associations may be caused by the relationship of both with diet. Frugivores tend to have larger home ranges and larger group sizes than folivores (Clutton-Brock and Harvey, 1977).

These multi-way relationships have been described as "syndromes" in which niche dimensions are correlated and their influence on brain size fluctuates according to their relative values (Nunn and van Schaik, 2002; Grueter, 2015). Indeed, the spatial mapping hypothesis (Clutton-Brock and Harvey, 1980; Milton, 1988) suggests that home range size affects brain size due to the cognitive demands of navigating a larger area, but also that the size of a range is linked to dietary demands (Deaner, Nunn and van Schaik, 2000). Home range size, diet and body size may form one such syndrome (Milton and May, 1976; Nunn and van Schaik, 2002). It is also possible that group size and home range size together represent a latent variable such as population density. This was suggested by Walker et al., who found that collapsing these variables in to one population density variable countered the problem of collinearity and uncovered clear relationships with life history variables (Walker *et al.*, 2006).

I found no support for the SBH as defined by Dunbar (Dunbar, 1992; Dunbar, 1998; Dunbar and Shultz, 2007a). Neocortex volume showed no relationship with group size in PGLS analysis across the three datasets when the rest of the variables were included, with or without the rest of brain as a covariate. Log likelihood ratio tests showed that its inclusion did not significantly improve the model's fit relative to a model comprising activity period and diet (Appendices 22 & 29). In Chapter 3, I found evidence to suggest that the Social Brain Hypothesis was only supported when using a specific dataset by Stephan and colleagues (1981). However, the results in the current chapter are partly based on the Stephan dataset, but do not show any associations which would support the SBH. It is likely that this is due to the inclusion of new data from Navarrete (pers. comm.) which has improved the dataset in terms of representation of important taxa and more up to date measurement methodology (issues with the original Stephan dataset are reviewed by Parker (Parker, 2015)). There is some evidence of a similar switching effect between diet and activity period in the neocortex models. Both are significant predictors in the body size controlled analyses, but never in the same model. Diet alone reaches significance in the RoB corrected condition, but model comparisons reveal that both improve the fit of a neocortex volume model in both body size and RoB corrected conditions. The Visual Brain Hypothesis (Barton, 1998) suggests that both diet and activity period are related to the size of the neocortex due to their individual relationships with visual adaptations (Barton, 1998). Therefore, these traits may also form a "syndrome" where their combined influence on neocortex volume is more potent than their individual effects.

4.4.3 A possible global brain size association with home range size

The pattern of switching between HRS and diet in body size corrected models of neocortex, cerebellum and cortex+cerebellum volume lend support to the possibility of their membership of a syndrome. This substitution pattern is not evident in the RoB corrected condition (Tables 4.3-4 – 4.3-6). This could possibly be due to functionality related to home range size being distributed across a number of brain structures, for which there is some evidence. In dataset 2 in the body size corrected condition, home range size is correlated with the volume of most brain structures, apart from the thalamus and the LGN. In the RoB corrected condition, home range size has no significant partial correlations with any structure apart from the striatum in dataset 3 (Table 4.3-6). This pattern suggests that home range size is not linked to the size of a particular structure independently of the rest of the brain (possibly apart from striatum); rather it is related more to global brain size. This potentially suggests that the cognitive functions associated with ranging are distributed widely across a number of structures, contrasting with the possibly more focal distribution of those associated with other behavioural-ecological correlates. The previous chapter demonstrated a strong effect of home range size on whole brain size emerging through two datasets. It was anticipated that structures implicated in route planning and locomotion, such as the cerebellum (MacLeod *et al.*, 2003; Cantalupo and Hopkins, 2010) and hippocampus (Hopkins, Lyn and Cantalupo, 2009) would show specific associations with home range, and that these associations might partly contribute to the whole brain size association. However, neither cerebellum volume nor hippocampus volume is correlated with HRS independently of other brain structures (Tables 4.3-4 – 4.3-6), contrary to predictions.

4.4.4 Disentangling the effects of home range size, body size and sexual dimorphism

The absence of any correlations with home range size in dataset 1 in the RoB corrected condition (Table 4.3-4) may be explained by the use of different body size estimates. Dataset 2 reported only female body size, whereas Dataset 1 averaged across the sexes apart from in sexually dimorphic species (size difference >10%) where only females were used. The correlation matrices (Appendices 36, 37 & 38) showed that home range size was more highly correlated with body size in the two datasets where males were included (datasets 1 and 3). Home range size was positively correlated with body size, as was sexual body size dimorphism (Cheverud, Dow and Leutenegger, 1985; Abouheif and Fairbairn, 1997; Smith and Cheverud, 1999; Borries *et al.*, 2013). Datasets 1 and 3 (which consist of mean measures from datasets 1 and 2) may therefore show increased variation in body size which may

obscure a relationship with home range size (which is correlated with body size) when it is included with it in a model.

The way sexual size dimorphism is accounted for in body size data could therefore have major implications for comparative work. Arguments have been made for using only female body size in order to overcome the potential confounding effects of size dimorphism, suggesting that the burden of maternal investment renders them more dependent on access to resources, making them the "ecological sex" (Nunn and van Schaik, 2002). It is interesting to note that the correlation of group size with a number of brain size metrics (relative brain size, neocortex volume, frontal lobe volume, etc.) was reported to be improved by using a female only sample (Dunbar and Shultz, 2017), which the authors suggested may indicate that female grouping patterns have driven primate brain evolution. Could the results of the current analyses on dataset 2 similarly suggest that female ranging has influenced primate brain evolution (as opposed to ranging in both sexes)? Possibly, but as we have not explicitly controlled for sex and there are a number of other potential sources of variation between the datasets, the evidence as yet does not allow us to speculate and a more prosaic explanation, such as the confounding effect of body size on home range size cannot be ruled out.

4.4.5 Cortico-cerebellar system

There is a tension between results shown in Chapter 2 which demonstrate the correlated evolution of the neocortex and cerebellum in agreement with the literature (Whiting and Barton, 2003; Barton, 2012) and the results of this chapter which fail to identify any behavioural-ecological correlates which might suggest functional independence of the cortico-cerebellar system. The analyses did not detect any clear or consistent correlations between the cortex+cerebellum measure and any of the behavioural-ecological variables. The inclusion of the rest of brain variable in the cortex+cerebellum models appears to remove all significant correlations that are present when RoB is not controlled for (apart from diet in dataset 1). The fact that the correlations between the volume of the cortex+cerebellum and both HRS and activity period are not present when the model includes RoB may suggest that the system is not wholly functionally isolated. However, the fact that the association between cortex+cerebellum and folivory persists (in dataset 1; Table 4.3-4) even when RoB is included in the model suggests that it would be premature to reject the notion of a complex that is (to some extent at least) functionally distinct from the rest of the brain.

The corticocerebellar complex also receives and sends projections from and to other brain structures and is comprised of more than just the cerebellum and the cortex. Input from the cortex reaches the cerebellum via the pontine nuclei and the cortex receives output from the cerebellum via the thalamus (Glickstein and Doron, no date; Whiting and Barton, 2003; Ramnani, 2006). Unfortunately, missing data and small sample size prohibited the inclusion of volumetric data from these structures in the corticocerebellar variable in this study without greatly reducing the sample size. Their inclusion might reveal a more functionally distinct system which can be demonstrated through correlations with behavioural-ecological variables. In addition, the corticocerebellar system may not be specialised for the types of functionality represented by the behavioural-ecological variables used in this study. The tension between previous results and those in this chapter may therefore be resolved by considering a different kind of selection pressure. The reciprocal loops between the two structures (Ramnani, 2006) have frequently been implicated in motor planning, but more recently has become associated with sequential processing necessary for extractive foraging, tool use (MacLeod *et al.*, 2003; Barton, 2012; Barton and Venditti, 2014) and even language (Leiner, Leiner and Dow, 1993). The predictor variables in this study did not represent this kind of function, which may be why no functional specialism was detected for this structural complex.

4.4.6 Limitations of the study

4.4.6.1 Overparameterisation

Overparameterisation is a potential problem for this study. While there is no hard and fast rule for the minimum number of cases per parameter, 10 is commonly used as a rule of thumb (Mundry, 2014). Taking in to account estimating lambda and the intercept, the total number of parameters for the largest models in this study is 8 (intercept, lambda, body size, rest of brain, activity period, diet, home range size and group size). The smallest sample size for a model of this size should therefore be 80. The largest dataset used in this study is dataset 3 which has a sample size of 62. The models presented above may therefore be characterised as overparamaterised which could be a factor in causing instability in results. Overparameterisation is addressed through either an increase in sample size or a decrease in parameter number. In this case, the sample used was the largest available while still using recent data obtained with modern techniques. The predictors used were those most germane to the question. Terrestriality was removed as it was not a significant predictor of brain size in any of the analyses in the previous chapter. Data reduction techniques such as principal

components analysis would have helped to reduce the number of predictors further but as these variables all form part of an animal's niche they are often linked (directly or indirectly) (Nunn and van Schaik, 2002) and so the specific biological syndromes represented by components may not be interpretable. Also, a PCA would not allow the clear examination of association between structures and specific behavioural-ecological variables. Testing these associations is necessary to test the predictions of the ecological, visual and social hypotheses of brain evolution, which was the aim of this chapter. Gathering more data (particularly neuroanatomical) to give a larger and more representative sample is the optimal way to ameliorate this issue.

4.4.6.2 Small, specialised clades with disproportionate influence

Another potential source of variation between datasets is the diversity of the primate order. Different clades can exhibit different patterns and rates of correlated evolution. For example, apes have been shown to break with the wider primate pattern of the close correlated evolution of neocortex and cerebellum, with ape cerebella undergoing accelerated expansion in comparison to neocortices (Barton and Venditti, 2014). Thus, a specialised clade which deviates from the overall pattern may introduce variation which can obscure relationships. Including clade as a factor in the models would ameliorate this issue, however due to the small number of extant ape species, even a comparative dataset which included every one would still not provide a sample large enough to avoid highly overparameterised models. The relationships between structure sizes and behavioural traits have also been shown to vary between clades. For example, Walker et al.'s 2006 findings suggested that home range size is a more important factor in brain size in Cercopithecoidea than in platyrrhines (Walker *et al.*, 2006).

4.4.6.3 System-level mosaic evolution may be critical to understanding the influence of behavioural ecology on the brain

Although there is good evidence to show that structures evolve in a mosaic fashion; changing size independently of one another, they are also highly interconnected and functions are highly distributed amongst them (Buckner and Krienen, 2013; Montgomery, Mundy and Barton, 2016). Therefore, a specific selection pressure may induce volumetric changes across a number of structures simultaneously. This may explain the apparent absence of relationships between neocortex volume and the behavioural-ecological variables included in this study. The neocortex is densely connected to other parts of the brain and is functionally heterogenous (Buckner and Krienen, 2013). Areas of the neocortex have also undergone their

own mosaic evolution (Barton, 2007). Groups of structures which form functional complexes like the corticocerebellar complex in this study may be usefully explored in future work.

4.4.7 Concluding remarks

The behavioural ecology of primate brain structures has been demonstrated to be complex, both in terms of the relationships between brain structures and behavioural correlates and between the structures themselves. Results are very sensitive to small differences between datasets as found in the analysis of whole brain size in the previous chapter. Possible causes of this instability range from the methodological: the sample size is too small, the collinearity of the predictors is too large; to the theoretical: the variables are not representative, the structures are still too functionally heterogeneous to detect a signal, or volume is too dissociated from actual computation and the premise that larger structure means more computation is faulty (Deacon, 1990; Chittka and Niven, 2009; Herculano-Houzel, 2009, 2010). Future work can take steps towards identifying the underlying causes and producing more stable results by developing larger comparative neuroanatomical datasets using modern imaging techniques to take more reliable measures, using functionally linked complexes of structures rather than the traditionally anatomically defined structures and using a more direct measure of computational capacity such as neuron number or density.

Despite the lack of stability in results, it is possible to draw a number of conclusions. The correlation between thalamus volume and group size is remarkably robust, but relatively unexplored in the literature. This, coupled with tantalising neuropsychological evidence for a link with sociality suggests more focused work specific to potential social functions of the thalamus is warranted. There is a potentially important relationship between home range size and brain and brain structure size that possibly confounds the relationship of the same with group size (Walker *et al.*, 2006) and thus should not be overlooked in comparative brain work. There is some signal of functional specialism for some structures, namely the thalamus, striatum, and lateral geniculate nucleus, but also evidence of both syndromes and the functional heterogeneity of individual structures, particularly in the model comparisons (Appendix 22 Appendix 35). Disentangling these effects from each other is not possible in the current analysis as regressions cannot reveal the direction of causality. Researchers are increasingly using path analysis in an effort to confront this problem (Lehmann, Korstjens and Dunbar, 2007; Dunbar and Shultz, 2007a; Hardenberg and Gonzalez-Voyer, 2013). Unfortunately, this technique cannot determine causal relationships as path analyses are a series of regression models, revealing only partial correlations which cannot tell us about the

direction of causality in a relationship (Denis and Legerski, 2003). Evidence for the functional specialism of the corticocerebellar complex was not found, but this may be due to the fact that the measure used did not represent the entire system.

Overall, the findings of this chapter have demonstrated that the behavioural-ecological correlates of brain structure sizes vary according to how size is controlled for and between different comparative datasets. The change in results when the volume of the rest of the brain is controlled for suggests that there is a degree of mosaic evolution occurring at the level of the neural system, as well as at the level of the individual structure. This chapter also underlines the difficulties of ascertaining the effects of individual behavioural-ecological traits when they all contribute to a common set of ecological niche dimensions and so are connected to one another, even if not always highly correlated. Ultimately, it is too simplistic to attribute a behaviour to the volume of an individual structure. As more comparative data on neuronal densities is published, we may begin to see more conclusive relationships emerging between behaviours and their neural substrates.

5 Life history correlates of primate brain structure volumes

5.1 Introduction

5.1.1 The role of ontogeny in primate brain evolution

While selective pressures like those examined in previous chapters have been shown to play a role in brain size and composition, these factors must interact with ontogenetic schedules that govern brain development. Brains that are large relative to body size take a long time to grow and reach maturity (Casey, Galvan and Hare, 2005; Barrickman *et al.*, 2008; Barton and Capellini, 2011). Large brained taxa like primates also tend to have longer lives (González-Lagos, Sol and Reader, 2010) and slower life histories with extended juvenile periods and later sexual maturity (Kaplan *et al.*, 2000; Charnov and Berrigan, 2005; Charvet and Finlay, 2012). This has led to a large body of comparative work examining the possible coevolution of these two traits (big brains and slow life histories), with mixed results (Barrickman *et al.*, 2008; Sol, 2009; van Woerden, van Schaik and Isler, 2010; Weisbecker *et al.*, 2015; Fristoe, Iwaniuk and Botero, 2017). Opinion is divided over whether this extension of life history is due to an elongated period of maternal investment in order to grow a larger brain (Martin, 1996; Isler and van Schaik, 2009), or whether a large brain promotes a longer life by providing the owner with increased behavioural flexibility to overcome life-limiting obstacles (Barrickman *et al.*, 2008; Sol, 2009). The two positions are not necessarily mutually exclusive.

Since the brain is composed of functionally and anatomically heterogeneous structures, it is possible that they vary independently according to either their developmental costs or the behavioural flexibility advantages they provide. Allman and colleagues presented evidence in support of this contention, reporting that structure volumes differentially correlated with lifespan and reproductive age (Allman, Mclaughlin and Hakeem, 1993). Their study, however, was not corrected for phylogeny, had a limited sample size and tested only two aspects of life history (lifespan and age at first reproduction). The present study examines the life history correlates of volumetric variation in primate structure sizes in more depth using appropriate comparative phylogenetic techniques.

5.1.2 Big, expensive brains: costs and benefits

5.1.2.1 Cost based hypotheses: the Maternal Energy Hypothesis & the Expensive Brain Hypothesis

Big brains are energetically costly (Aiello and Wheeler, 1995; Isler and van Schaik, 2009) and so require a great deal of metabolic investment. Since most of brain growth (in volumetric terms) occurs prenatally and during infanthood (Leigh, 2004), the energetic costs are met by the mother during this time. Cost based hypotheses assert that life history correlates of brain size reflect the duration of brain growth. Martin (1981, 1996), proffered an early incarnation of such a hypothesis, suggesting that the basal metabolic rate (BMR) of the mother determines the energy available for the offspring during gestation and lactation and so dictates its brain growth. This "Maternal Energy Hypothesis" therefore predicts that mammalian brains grow as large as allowed by the constraints of gestation and lactation duration, which are governed by maternal BMR. Martin also suggested that since these developmental constraints govern brain size, behavioural-ecological correlates of relative brain size do not reflect direct selection on cognitive specialisations.

The Maternal Energy Hypothesis has been both supported and contradicted by various work since (Isler and van Schaik, 2009; Barton and Capellini, 2011). Recent work suggests that maternal BMR does have a (weak) prenatal effect on offspring brain size (Barton and Capellini, 2011). However, maternal investment in terms of energy provision to offspring occurs both pre and postnatally. During gestation, energy is provided directly from the maternal circulation to the foetus via the placenta. Postnatally, neonatal energy must be sourced via the digestion of maternal milk. While both represent maternal energetic investment, the energy transfer systems are quite different in terms of how directly available energy is to the infant. Consequently, they appear to be subject to different selective pressures, varying between altricial and precocial species. The Expensive Brain Hypothesis (Isler and van Schaik, 2009) is a later expansion of the Maternal Energy Hypothesis. It suggested that brain size in multiparous precocial mammal species was reported to be positively correlated with gestation and lactation, with a decrease in annual fertility (offspring or litters per year) to balance the metabolic cost, Multiparous altricial mammals differed, however, as gestation length was not related to brain size, rather litter size was reduced to provide more maternal energy per offspring (Isler and van Schaik, 2009) (this latter finding was subsequently contradicted by Barton and Capellini (2011)). The timing of the point of cessation of gestation and commencement of lactation within the wider maternal investment

period appears to be influenced by different factors to those that govern the overall duration of maternal investment (Dubman, Collard and Mooers, 2012). The Maternal Energy and the Expensive Brain hypotheses explain this apparent dual system by suggesting that brain size can be modified by selection on both maternal BMR and the duration of maternal investment. This model helps to resolve apparently paradoxical findings such as the delayed prenatal neurodevelopment in precocial mammals (Workman *et al.*, 2013).

5.1.2.2 Benefit based hypotheses: The Cognitive Buffer Hypothesis

The Cognitive Buffer Hypothesis (hereafter "CBH") explains life-history correlates of brain size from a different perspective. It emerged primarily from the finding that large brains are associated with longer lives (Sacher, 1959; Allman, Mclaughlin and Hakeem, 1993; González-Lagos, Sol and Reader, 2010) as well as slower life histories overall (Kaplan *et al.*, 2000; Charnov and Berrigan, 2005; Charvet and Finlay, 2012). Developing a large brain is costly in terms of delayed reproduction (both in terms of individuals taking longer to reach sexual maturity and in terms of mothers having longer interbirth intervals). This fitness cost should be balanced by a fitness benefit in order to be selected (González-Lagos, Sol and Reader, 2010). There are two variants of the CBH. The original iteration argues that larger brains bestow adaptive benefits on their owners in the form of behavioural flexibility which reduces extrinsic mortality by enabling the animal to behaviourally adapt to novelty or complexity in its environment, promoting longevity by "enhancing survivability" (Allman, Mclaughlin and Hakeem, 1993, p. 3562). In terms of the specific mechanism by which fitness is increased, some comparative biologists suggest that larger brained animals benefit from a longer reproductive life (adulthood) which balances the costs of delayed reproduction necessary for development of a large brain and the risks associated with being a subadult for a long period of time, such as increased chance of infant/juvenile mortality (Barrickman *et al.*, 2008; Sol, 2009; González-Lagos, Sol and Reader, 2010). This variant of the CBH is hereafter referred to as the "longevity payoff CBH".

Longer life histories extend juvenile periods as well as reproductive lifespans. Some biologists have emphasised the importance of this long juvenile period, which affords increased opportunity for social learning as a subadult (Ross and Jones, 1999), thus likely extending the reproductive lifespan (Barrickman *et al.*, 2008). This variant of the CBH is hereafter referred to as "juvenile learning payoff CBH". The two interpretations are not mutually exclusive; rather they differ over whether the time required to learn skills which "buffer" against mortality or the time available for deployment of those skills is the most

important force in shaping post weaning life history. It has been demonstrated that social learning (based on a measure which counted reported instances of this behaviour in the literature, corrected for a measure of research effort (Reader, Hager and Laland, 2011) correlates with both brain volume and reproductive lifespan, supporting the idea that a larger brain supports increased rates of social learning, which in turn promotes a longer reproductive life or adulthood (Street *et al.*, 2017). In this scenario, both interpretations are equally valid.

5.1.3 Developmental scheduling in the mosaic brain

The hypotheses discussed so far relate to whole brain size. However, brain structures show a degree of heterochronicity in their development (Huttenlocher and Dabholkar, 1997; Charvet and Finlay, 2012; Workman *et al.*, 2013; Sherwood and Omez-Robles, 2017). While some large-scale patterns suggest an overarching size-dependent scaling in brain development (Finlay and Darlington, 1995; Clark, Mitra and Wang, 2001; Yopak *et al.*, 2010), there is variation in the scaling of individual structures that appears to be a) independent of these global scaling factors and b) associated with selection on function. It has also been demonstrated that structures' developmental scheduling is likely scheduled by structure-specific genes rather than genes coordinating global brain growth (Harrison and Montgomery, 2017).

Literature describing developmental growth of individual brain regions in non-humans or volumetric changes spanning birth in any taxon is sparse. There are some indications of broad scale patterns that are shared between primate species; for example, the brain of the brown capuchin (*Cebus apella*) is reported to show a rapid increase in total brain volume and specifically white matter volume early in postnatal life, similar to humans (Phillips and Sherwood, 2008). However, this study also reports differences between primate taxa in terms of brain maturity at birth, with rhesus macaques' (*Macaca mulatta*) neonatal brain volume a larger percentage of their adult volume in comparison to capuchins and chimpanzees (p. 661).

The growth of different brain structures varies during gestation and infancy. An early study examining these patterns in *Macaca nemestrina* demonstrated that most of the brain undergoes roughly linear volumetric growth over much of the gestation period. This contrasted with the cerebellum and diencephalon which had a relatively slow rate of early growth, accelerating later in gestation (DeVito, Graham and Sackett, 1989). Postnatally, there is a pronounced divergence in the rates and even direction of volumetric change in the gross

structures. The diencephalon, mesencephalon, pons and medulla all either plateaued or decreased in volume after birth. In contrast, the cerebellum and telencephalon continued to gain volume, with the cerebellum continuing to grow once telencephalic growth had begun to level out (DeVito *et al.*, 1986).

The development of the cerebellum in humans is notably distinctive; appearing to follow a quite protracted growth trajectory compared to other gross structures. It undergoes a large amount of postnatal volumetric growth, growing by around 240% in absolute size in the first year postnatally, while the cerebral hemispheres show a more modest ~87% increase (Knickmeyer et al., 2008). Other subcortical regions are also increasing in volume during this time but to a smaller degree than the cerebellum (around 132%). Indeed, the largest proportion of growth in the cerebellum occurs postnatally, within the first two years (Wu, Chen and Shen, 2011). This is in part due to the postnatal proliferation of cerebellar granule cells; 85% of which are generated between birth and one year (Kiessling *et al.*, 2014). The cerebellum attains its peak volume at around 13.5 years (females peak earlier than males; 11.8 and 15.5 years respectively), later than the cerebrum (Tiemeier *et al.*, 2010). It has been suggested that it is amongst the latest of the structures to attain peak volume (Giedd, Schmitt and Neale, 2007). Later juvenile cerebellar growth (post 8 years) is mostly attributable to increasing white matter volume (Ostby *et al.*, 2009). There is some evidence to suggest that primates have relatively large cerebella in comparison to other mammalian taxa (Yopak *et al.*, 2010; Barton, 2012), and ape cerebella are larger than those of non-apes (Rilling and Insel, 1998; Marino *et al.*, 2000; Barton and Venditti, 2014). Apes also have extended juvenile periods (Kelley, 2004), pointing to a potentially revealing link between this and their large, late maturing cerebella.

In a study of volumetric changes in humans aged 8 – 30 years, the cerebral cortex and components of the striatum (pallidum, putamen and accumbens area) showed the largest decrease in volume across this age range (Ostby *et al.*, 2009). The hippocampus shows no significant size changes during the second postnatal year (the earliest it can be reliably isolated using MRI) (Knickmeyer *et al.*, 2008) and does not appear to vary significantly in size later in life (Ostby *et al.*, 2009).

5.1.4 Questions and predictions addressed in the current study

In order to further examine the relationship between brain evolution, development and life history, the present study examines life history correlates of brain structure sizes; testing

predictions arising from both cost and benefit based hypotheses. Can variation in brain structure sizes be attributed to developmental costs or the buffering benefits they provide, or both? The different hypotheses regarding the relationship between primates' elongated life histories and their large brains make different predictions with regard to how the size of individual structures relate to specific life history traits. The CBH would predict a correlation between structure volumes and lifespan. However, this does not discount a role of developmental costs. If structures also correlate with maternal investment and do not correlate with lifespan or fail to correlate with it once maternal investment is controlled for, then it suggests that the relationship between lifespan and structure size is a product of the costs of development (Barton and Capellini, 2011). If a positive association with longevity persists after maternal investment is included, it suggests that the increased size of the structure confers some adaptive advantage which buffers against mortality. If the reproductive lifespan is correlated with structure volume, then it would support the hypothesis that larger brain components evolved due to the benefits of the learned skills which prolong life. As outlined above, the juvenile learning payoff CBH would suggest that it is the length of the period during which the skills are learned that should show a relationship with size. Therefore, if juvenile period correlates with a structure size independently of maternal investment, it would support the juvenile learning payoff CBH. Examining the relationships between structure volumes and life history traits should reveal whether the mosaic growth of individual structures can be explained in terms of developmental costs or cognitive buffering benefits. The predicted associations between structures and life history traits are detailed below.

5.1.4.1 Neocortex and hippocampus volume predicted to correlate with prenatal maternal investment

Based on the developmental scheduling of structure growth as described above, I derived a set of predictions regarding the life history correlates of adult structure volumes. Neocortex and hippocampus volumes were predicted to follow the predictions of the developmental costs hypotheses, correlating with prenatal maternal investment due to a larger proportion of their growth occurring prenatally. Since all brain structures grow prenatally (to varying extents), one might expect all brain structure volumes to correlate with the duration of prenatal investment. However, since prenatal and postnatal maternal investment duration are positively correlated, when both are included in a model it is likely that only the measure which is most strongly related to the structure volume in question will correlate.

5.1.4.2 Striatum and cerebellum volume predicted to correlate with postnatal maternal investment

Postnatal brain growth is positively associated with lactation duration independently of gestation duration (Barton and Capellini, 2011). It is therefore reasonable to predict that volumes of individual structures which undergo significant postnatal growth will show a specific relationship with postnatal maternal investment (lactation). Therefore, cerebellum and striatum volume were predicted to correlate with postnatal maternal investment due to the demonstrated postnatal expansion of the cerebellum and caudate nucleus (a component of the striatum).

5.1.4.3 Cerebellum volume predicted to correlate with duration of juvenile period

Cerebellum volume was additionally predicted to correlate with juvenile period as the cerebellum continues to increase in volume well in to juvenility, in humans at least (Wu, Chen and Shen, 2011) (post lactation, pre first parturition). If juvenile period is correlated with cerebellum volume independently of the duration of maternal investment, this would suggest that postponing cerebellar maturation is an adaptive trait, rather than a by-product of the extension of life history due to developmental costs. The late maturation of the cerebellum might indicate that environmental exposure is important for its development. Cerebellum volume was therefore predicted to support predictions of both developmental costs hypotheses and the CBH.

5.1.4.4 Cerebellar associations with postnatal life history variables predicted to be particularly associated with the apes

As detailed above, cerebellum volume is particularly large in the apes (Barton and Venditti, 2014). Two predictions are derived from this observation: a) if found, a correlation between relative cerebellum volume and lactation or juvenile period may be particularly associated with the apes, so that the correlation is not present when apes are removed from the analysis and b) that lactation duration (relative to body size) might be elongated in the apes.

5.2 Methods

5.2.1 Brain structure variables

The brain structures chosen for analyses were neocortex, cerebellum, hippocampus and striatum. These four structures were included in a study by Knickmeyer and colleagues (Knickmeyer *et al.*, 2008) which provided a longitudinal comparison of the volumetric development of several human brain structures from birth to two years: a stage in which there is reportedly divergence in structural growth trajectories (DeVito *et al.*, 1986). This study therefore presented clear comparisons between structure volumes over time using a single method (MRI), which allowed clear predictions about their life history correlates to be derived. Brain volume was also analysed to allow comparison with previous work addressing the role of life history in brain size which has primarily investigated the whole brain. It also gives context to structure analyses in terms of how relationships between structure sizes and life history may be related to brain size.

5.2.2 Life history variables

Life history data were taken from Pantheria (Jones *et al.*, 2009): a large online repository of physical and behavioural data for a wide range of mammalian taxa. The variables extracted for analysis were gestation duration (days), weaning age (days), age at first parturition (days) and maximum longevity (months). Some variables were then further modified for analysis. In addition to being analysed separately, gestation and lactation periods were summed to create an overall measure of duration of maternal investment. Juvenile period was defined as the period between weaning and age at first parturition. This was operationalised by subtracting weaning age from age at first parturition. Age at first parturition was subtracted from maximum longevity to give a measure of reproductive lifespan. Post-infancy period combined juvenile period and reproductive lifespan.

Gestation and weaning age (hereafter referred to as lactation duration) were included to give measures of pre and postnatal maternal investment. Juvenile period is particularly relevant to the juvenile learning payoff CBH, which proposes that the elongation of this phase facilitates the learning of complex skills. Age at first parturition represents the beginning of adulthood. This period is of importance to this study as it represents the cessation of dependence and the period where skills learned socially during juvenility may be implemented (Kaplan *et al.*, 2000; Street *et al.*, 2017). The composite variables maternal investment period and post-infancy period give more global measures. These were particularly relevant for testing for

effects of the totality of maternal investment as the total duration of investment may be more relevant to structure growth than whether it is pre or postnatal. The life history variables are nested within each other, starting with overall lifespan, then dividing into duration of maternal investment and duration of post-infancy period, and finally dividing maternal investment into pre and postnatal investment (gestation and lactation) and post-infancy into juvenile period and reproductive period. Figure 5:a below illustrates this hierarchy.

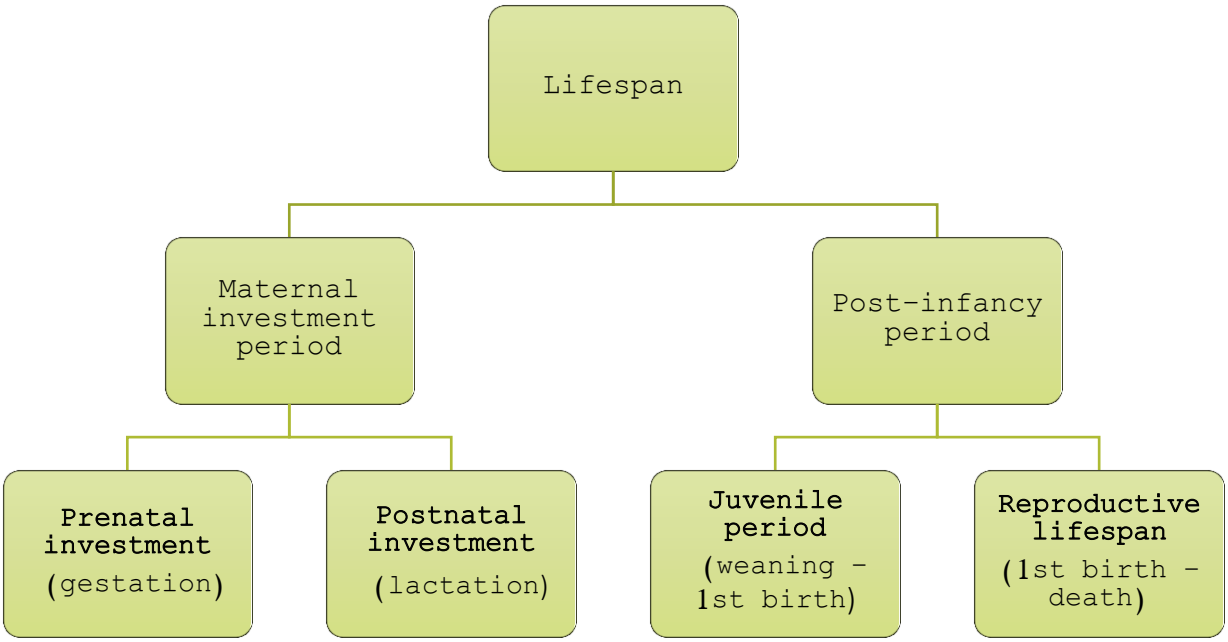


Figure 5:a - Hierarchical organisation of predictor variables

5.2.3 Statistical analyses

Phylogenetic least squares (PGLS) regressions were employed to test for correlated evolution between the life history predictors and structure volumes. λ was estimated by maximum likelihood and the consensus phylogeny from 10k trees (Arnold, Matthews and Nunn, 2010) was used. All continuous variables were log10 transformed to satisfy assumptions of normality, and all PGLS models controlled for body mass by including it as a predictor to control for its effect on structure volumes (Smith, 1999; Garcia-Berthou, 2001; Freckleton, 2002). PGLS models were also compared using log likelihood ratio tests and Akaike's Information Criterion (AIC) (Akaike, 1974) to determine which variables significantly improved fit relative to a purely allometric model (body size as the only predictor). As discussed in previous chapters, both methods of model comparison are included as they offer the opportunity for slightly different interpretation. Log likelihood ratio tests compare a

model to a null model, giving a measure of absolute fit. The AIC on the other hand compares models relative to each other, so that if all models constitute a poor fit, this is not apparent when only AIC is used (Maydeu-Olivares and García-Forero, 2010).

Following Barton and Capellini (2011), gestation length was controlled for (in addition to body size,) in the postnatal investment model to distinguish between the two phases of maternal investment and ensure that any effect found was independent of prenatal investment and so specific to the postnatal phase of investment. Juvenile period was controlled for (in addition to body size) in the models examining the effect of reproductive period to distinguish whether any effect found was independent of juvenile period or whether it could be attributed to the duration of overall post-infancy, possibly representing a period of feeding independence. Inspection of the variance inflation factors (VIFs) suggests that reproductive period and post infancy period are highly collinear with longevity, with values in excess of 173 in the latter. This is unsurprising as the post infancy period comprises the majority of the overall lifespan. Longevity therefore cannot be included in models which included these two variables. VIFs for all other variables in any of the combinations used in the experimental models are less than 3.01 and so collinearity is not deemed to be a problem (Garamszegi, 2014).

To assess the potential influence of the difference between apes and other primates on results, the apes (n=4) were removed from the dataset and the analyses rerun. Phylogenetic ANCOVA was employed to test for a significant difference between apes and non-apes in lactation duration accounting for body size. Relative gestation duration was also analysed with this method to ascertain whether there was a wider change in overall maternal investment in this taxon, rather than an independent change in lactation. Models with same and different slopes for each taxon were compared using Akaike's Information Criterion (AIC) (Akaike, 1974) to determine which provided the best fit. Data were again sourced from Pantheria (Jones *et al.,* 2009). As there was better data availability for gestation and lactation duration than the other variables in the main analysis, it was possible to assemble larger samples for the ANCOVA analyses. For gestation, the dataset comprised 146 primate species, of which 13 were apes. For lactation, the dataset comprised 119 primate species, of which 10 were apes.

5.3 Results

5.3.1 Relative brain size

Relative whole brain volume (brain size controlled for by the addition of body size as a covariate in PGLS) correlated with lifespan (t_{43}=2.11, p<0.05), but when maternal investment period was included, longevity no longer reached significance (Table 5.3-1). Longer periods of maternal investment were positively associated with relatively larger brains, controlling for longevity and body mass (t_{43}=2.7, p<0.01). Post-infancy lifespan was not a significant predictor of relative brain size (t_{43}=1.89, p=0.07). Exploring the maternal investment period in more detail, gestation period was positively correlated with brain size (t_{43}=2.07, p<0.05) independently of the relationship between longevity and brain size, which remained significant. However, when lactation was included neither it nor gestation reached significance, and longevity also fell below the threshold. Log likelihood ratio tests revealed that the inclusion of both gestation and lactation improved a model of longevity only and longevity + gestation respectively. Neither juvenile period nor reproductive period (controlling for juvenile period in the latter) was correlated with brain volume, however juvenile period did improve the fit over an allometric model. Model comparisons using AIC (Table 5.3-1) showed that the postnatal maternal investment model (gestation plus lactation duration including body size as covariate) provided the best and most parsimonious fit.

5.3.2 Neocortex volume

Longevity was not correlated with neocortex volume; however, the duration of maternal investment showed a significant positive association (t_{43}=2.56, p<0.05) (Table 5.3-3). Post infancy lifespan was not a significant predictor of neocortex volume. Gestation showed a significant correlation, but this association failed to reach significance when lactation was included in the model, indicating its relationship with neocortex volume was not independent of lactation duration. Lactation was not a significant predictor. A log likelihood ratio test showed that gestation significantly improved the fit of a purely allometric (body size only) model, but the addition of lactation did not improve the fit further (Table 5.3-4). There was a positive association with juvenility, but this was non-significant after the inclusion of reproductive period in the model. A log likelihood ratio test showed that juvenile period significantly improved fit over an allometric model, but the inclusion of reproductive period did not improve fit. Whilst the postnatal maternal investment (lactation) model had the lowest AIC value, prenatal maternal investment, juvenility and reproductive lifespan models showed

a comparable fit to the data (Table 5.3-3). As the prenatal maternal investment and juvenility models were equally parsimonious, one cannot be supported over the other.

5.3.3 Cerebellum volume

Cerebellum volume did not show an association with lifespan (Table 5.3-5). Maternal investment had a significant positive relationship with cerebellum volume (t_{43}=3.52, p<0.01), but post infancy lifespan showed no such association (t_{43}=1.3, p=0.2). Gestation duration was positively correlated with cerebellum volume, but this relationship ceased to reach significance when lactation was included in the model. As predicted, lactation predicted cerebellum volume (t_{42}=2.48, p<0.05) independently of gestation duration. Juvenile period duration also showed a significant positive association (t_{42}=2.6, p<0.05) which was independent of reproductive lifespan. Log likelihood ratio testing further demonstrated that reproductive lifespan did not improve fit (Table 5.3-6). Model comparisons using AIC values showed that postnatal maternal investment and juvenile period models (separately) had the best support (Table 5.3-5).

5.3.4 Hippocampus volume

Lifespan was not a significant predictor of hippocampus volume, and neither maternal investment duration or length of post-infancy period showed a significant correlation either (Table 5.3-7). The AIC values of the two models were virtually identical (maternal investment AIC=-20.52, post-infancy AIC=-20.5) and so neither could be deemed a better fit than the other. Prenatal investment, postnatal investment, juvenile period duration and reproductive lifespan were not correlated with hippocampus volume and log likelihood ratio tests showed that none of these variables improved fit over a purely allometric model (Table 5.3-8). AIC values showed that no model had better support than the allometric (body size only) model.

5.3.5 Striatum volume

Lifespan was also not a significant predictor of striatum volume (Table 5.3-9). The duration of maternal investment was significantly associated with striatum volume (t_{43}=2.38, p<0.05), post-infancy period was not. Gestation was correlated with striatum volume independently of lactation. Log likelihood ratio tests showed that including lactation in a body size + gestation model did not significantly improve the fit of the model. Juvenile period showed a significant correlation when it was the sole predictor (along with the covariate body size) but was non-significant when reproductive period was included (Table 5.3-10). Comparison of AIC values

indicated that the pre and postnatal investment models were equally supported as the best model.

5.3.6 Taxonomic differences between apes and non-apes

Hominoidea (n=4 with data on both lactation and gestation length) were removed from the sample to examine the effect their absence might have on results. When the PGLS analyses was rerun without the apes, the association between lactation and cerebellum volume was no longer present (t_{38}=1.6, p=0.12). Similarly, juvenile period was no longer a significant predictor. AIC values showed that no model had better support than the purely allometric model. For comparative purposes, the PGLS analyses on hippocampus volume were also rerun. The hippocampus was chosen for this purpose as the neocortex, striatum and cerebellum are part of an anatomically and functionally integrated network (Bostan, Dum and Strick, 2010) which also makes up a large proportion of total brain size. The hippocampus results were not qualitatively changed by the removal of the apes.

The results of the phylogenetic ANCOVA on lactation duration showed some evidence of a difference between apes and non-apes (Table 5.3-11). The different slopes model had the lowest AIC value, but could not be separated from the same slopes models as the AIC difference was less than 2 (1.69). The different slopes model showed that the slope for apes was significantly different to that for non-apes, but as the model projects beyond the range of the ape data it would be unwise to attribute much significance to this. The phylogenetic ANCOVA on gestation duration showed no evidence of any difference between the apes and the non-apes in either the same slopes or different slopes model (Table 5.3-12). The ape-non-ape difference did not reach significance in either case, and there was no interaction with body size.

Table 5.3-1 - PGLS analysis of the life history correlates of brain volume

	Lifespan	Total maternal investment duration	Post-infancy lifespan	Prenatal maternal investment	Postnatal maternal investment	Juvenility	Adulthood
	t_{43} *(p)*	t_{42} *(p)*	t_{43} *(p)*	t_{42} *(p)*	t_{41} *(p)*	t_{42} *(p)*	t_{41} *(p)*
Intercept	**4.4 (<0.001‡)**	**2.98 (<0.01†)**	-0.52 (0.6)	0.95 (0.35)	1.34 (0.19)	**2.76 (<0.01†)**	1.78 (0.08)
Body Mass	**15.38 (<0.001‡)**	**11.59 (<0.001‡)**	**15.85 (<0.001‡)**	**11.32 (0.001‡)**	**10.08 (<0.001‡)**	**12.11 (<0.001‡)**	**11.4 (<0.001‡)**
Maternal investment	-	**2.7 (<0.01†)**	-	-	-	-	-
Post-weaning	-	-	1.89 (0.07)	-	-	-	-
Longevity	**2.11 (<0.05*)**	1.68 (0.1)	-	**2.06 (<0.05*)**	1.76 (0.09)	1.72 (0.09)	
Gestation	-	-	-	**2.07 (<0.05*)**	1.51 (0.14)	-	-
Lactation	-	-	-	-	1.99 (0.05)	-	-
Juvenile period	-	-	-	-	-	1.59 (0.12)	1.94 (0.06)
Reproductive lifespan	-	-	-	-	-	-	1.6 (1.12)
Lambda	.7	.57		.62	.6	.59	.57
R^2	.9	.92		.91	.92	.91	.91
AIC model comparison	-64.68	-	-	-67.05	-69.3 (AIC_{min})	-65.2	-64.8

Variables not included in models are indicated with a dash (-). Longevity is included in these models as it had a significant association with brain volume. Longevity was not included in post infancy lifespan and adulthood models due to high VIFs. Degrees of freedom are indicated in subscript after "*t*".
Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01. ‡ = p <0.001

Table 5.3-2 -Log likelihood ratio test of life history models of brain volume

	Predictors (response variable = brain volume)	Log likelihood	χ^2	*p*
Maternal investment models	Body size	33.11		
	Body size + longevity	35.34	4.47	**<0.05***
	Body size + longevity + gestation	37.52	4.37	**<0.05***
	Body size + longevity + gestation + lactation	39.65	4.25	**<0.05***
Post weaning models[a]	Body size + juvenile period	35.01	3.99	**<0.05***
	Body size + juvenile period + reproductive period	36.4	2.6	0.11

[a]Longevity could not be included in the post weaning models due to high VIFs. **Bold** denotes significance at at least the α<0.05 level. * = p <0.05

Table 5.3-3 - PGLS analysis of the life history correlates of neocortex volume

	Lifespan	Total maternal investment duration	Post-infancy lifespan	Prenatal maternal investment	Postnatal maternal investment	Juvenility	Adulthood
	t_{43} *(p)*	t_{43} *(p)*	t_{43} *(p)*	t_{43} *(p)*	t_{42} *(p)*	t_{43} *(p)*	t_{42} *(p)*
Intercept	**1.91 (<0.05*)**	**3.774 (<0.001‡)**	-0.82 (0.41)	0.83 (0.41)	1.04 (0.3)	**2.63 (<0.05*)**	0.4 (0.69)
Body Mass	**12.27 (<0.001‡)**	**10.18 (<0.001‡)**	**12.68 (<0.001‡)**	**11.03 (<0.001‡)**	**8.83 (<0.001‡)**	**10.67 (<0.001‡)**	**9.62 (<0.001‡)**
Maternal investment	-	**2.56 (<0.05*)**	-	-	-	-	-
Post-weaning	-	-	1.76 (0.09)	-	-	-	-
Longevity	1.91 (0.06)	-	-	-	-	-	-
Gestation	-	-	-	**2.26 (<0.05*)**	1.72 (0.09)	-	-
Lactation	-	-	-	-	1.61 (0.12)	-	-
Juvenile period	-		-	-	-	**2.1 (<0.05*)**	1.98 (0.05)
Reproductive lifespan	-	-	-	-	-	-	1.42 (0.16)
Lambda	.70	.48	.69	.51	0.52	.47	.56
R^2	.86	.89	.85	.88	.89	.89	.88
AIC model comparison	-43.54	-	-	-44.88	-45.62 (AIC_{min})	-44.05	-44.1

Variables not included in models are indicated with a dash (-). Degrees of freedom are indicated in subscript after "*t*".
Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01. ‡ = p <0.001

Table 5.3-4 - Log likelihood ratio test of life history models of neocortex volume

	Predictors (response variable = brain volume)	Log likelihood	χ^2	*p*
Maternal investment models	Body size	22.94		
	Body size + gestation	25.44	5	**<0.05***
	Body size + gestation + lactation	26.81	2.75	0.1
Post weaning models	Body size + juvenile period	25.03	4.18	**<0.05***
	Body size + juvenile period + reproductive period	26.05	2.05	0.15

Bold denotes significance at at least the α<0.05 level. * = p <0.05

Table 5.3-5 - PGLS analysis of the life history correlates of cerebellum volume

	Lifespan	**Total maternal investment duration**	**Post-infancy lifespan**	**Prenatal maternal investment**	**Postnatal maternal investment**	**Juvenility**	**Adulthood**
	t_{43} *(p)*	t_{43} *(p)*	t_{43} *(p)*	t_{43} *(p)*	t_{42} *(p)*	t_{43} *(p)*	t_{42} *(p)*
Intercept	1.66 (0.1)	**-2.38 (<0.05*)**	-0.65 (0.52)	1 (0.32)	1.47 (0.15)	1.44 (0.16)	-0.05 (0.96)
Body Mass	**20.27 (<0.001‡)**	**16.64 (<0.001‡)**	**21.42 (<0.001‡)**	**23.22 (<0.001‡)**	**15.25 (<0.001‡)**	**15.73 (<0.001‡)**	**14.27 (<0.001‡)**
Maternal investment	-	**3.52 (<0.01†)**	-	-	-	-	-
Post-weaning	-	-	1.3 (0.2)	-	-	-	-
Longevity	1.58 (0.12)	-	-	-	-	-	-
Gestation	-	-	-	**2.13 (<0.05*)**	1.19 (0.24)	-	-
Lactation	-	-	-	-	**2.48 (<0.05*)**	-	-
Juvenile period	-	-	-	-	-	**2.65 (<0.05*)**	**2.6 (<0.05*)**
Reproductive lifespan	-	-	-	-	-	-	0.92 (0.36)
Lambda	.00	.00	.00	.00	.00	.00	.00
R^2	.96	.97	.96	.96	.97	.97	.97
AIC model comparison	-67.44	-	-	-69.45	-73.73 (AIC_{min})	71.83	-70.74

Variables not included in models are indicated with a dash (-). Degrees of freedom are indicated in subscript after "*t*".
Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01, ‡ = p <0.001

Table 5.3-6 - Loglikelihood ratio tests of life history models of cerebellum volume

	Predictors (response variable = brain volume)	Log likelihood	χ^2	*p*
Maternal investment models	Body size	35.43		
	Body size + gestation	37.73	4.6	<0.05*
	Body size + gestation + lactation	40.87	6.28	<0.05*
Post weaning models	Body size + juvenile period	38.91	6.98	<0.01†
	Body size + juvenile period + reproductive period	39.37	0.91	0.34

Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01

Table 5.3-7 - PGLS analysis of the life history correlates of hippocampus volume

	Lifespan	Total maternal investment duration	Post-infancy lifespan	Prenatal maternal investment	Postnatal maternal investment	Juvenility	Adulthood
	t_{43} *(p value)*	t_{43} *(p value)*	t_{43} *(p value)*	t_{43} *(p value)*	t_{42} *(p value)*	t_{43} *(p value)*	t_{42} *(p value*
Intercept	**2.14 (<0.05*)**	**-2.31 (<0.05*)**	-0.65 (0.52)	0.01 (1)	0.05 (0.96)	**2.05 (<0.05*)**	1.42 (0.16)
Body Mass	**8.69(<0.001‡)**	**6.77 (<0.001‡)**	**8.92 (<0.001‡)**	**6.78 (<0.001‡)**	**5.98 (<0.001‡)**	**7.23 (<0.001‡)**	**6.85 (<0.001‡)**
Maternal investment	-	0.31 (0.76)	-	-	-	-	-
Post-weaning	-	-	-0.27 (0.79)	-	-	-	-
Longevity	-0.33 (0.75)	-	-	-	-	-	-
Gestation	-	-	-	1.39 (0.17)	1.46 (0.15)	-	-
Lactation	-	-	-	-	-0.49 (0.63)	-	-
Juvenile period	-	-	-	-	-	-0.04 (0.96)	-0.03 (0.98)
Reproductive lifespan	-	-	-	-	-	-	-0.36 (0.73)
Lambda	.46	.45	.46	.45	.45	.45	.46
R^2	.72	.73	.72	.74	.73	.72	.72
AIC model comparison	-20.53	-	-	-22.45 (AIC_{min})	-20.71	-20.42	-18.56

Variables not included in models are indicated with a dash (-). Degrees of freedom are indicated in subscript after "*t*".
Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01. ‡ = p <0.001

Table 5.3-8 - Log likelihood ratio tests of life history models of hippocampus volume

	Predictors (response variable = brain volume)	Log likelihood	χ^2	*p*
Maternal investment models	Body size	13.21		
	Body size + gestation	14.25	2.03	0.15
	Body size + gestation + lactation	14.35	0.26	0.61
Post weaning models	Body size + juvenile period	13.21	0.00	0.96
	Body size + juvenile period + reproductive period	13.28	0.14	0.71

Bold denotes significance at at least the α<0.05 level. * = p <0.05

Table 5.3-9 - PGLS analysis of the life history correlates of striatum volume

	Lifespan	Total maternal investment duration	Post-infancy lifespan	Prenatal maternal investment	Postnatal maternal investment	Juvenility	Adulthood
	t_{43} *(p)*	t_{43} *(p)*	t_{43} *(p)*	t_{43} *(p)*	t_{42} *(p)*	t_{43} *(p)*	t_{42} *(p)*
Intercept	0.78 (0.44)	0.96 (0.34)	-0.94 (0.35)	-1.07 (0.29)	-0.9 (0.37)	0.07 (0.94)	-0.58 (0.56)
Body Mass	**11.77 (<0.001‡)**	**11.31 (<0.001‡)**	**12.12 (<0.001‡)**	**16.67 (<0.001‡)**	**10.9 (<0.001‡)**	**10.83 (<0.001‡)**	**9.29 (<0.001‡)**
Maternal investment	-	**2.38 (<0.05*)**	-	-	-	-	-
Post-weaning	-	-	1.41 (0.17)	-	-	-	-
Longevity	1.47 (0.15)	-	-	-	-	-	-
Gestation	-	-	-	**3.08 (<0.01†)**	**2.54 (<0.05*)**	-	-
Lactation	-	-	-	-	0.87 (0.39)	-	-
Juvenile period	-		-	-	-	**2.38 (<0.05*)**	2 (0.05)
Reproductive lifespan	-		-	-	-	-	0.98 (0.33)
Lambda	.77	.00	.77	.00	.00	.00	.65
R^2	.83	.93	.83	.94	.93	.93	.86
AIC model comparison	-44.2	-	-	-51.31 (AIC_{min})	-50.13	-47.85	-45.44

Variables not included in models are indicated with a dash (-). Degrees of freedom are indicated in subscript after "*t*".
Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01, ‡ = p <0.001

Table 5.3-10 - Log likelihood ratio tests of life history models of striatum volume

	Predictors (response variable = brain volume)	Log likelihood	χ^2	*p*
Maternal investment models	Body size	24.1		
	Body size + gestation	28.66	9.12	**<0.01†**
	Body size + gestation + lactation	29.07	0.82	0.37
Post weaning models	Body size + juvenile period	26.93	5.66	**<0.05***
	Body size + juvenile period + reproductive period	26.72	0.41	0.52

Bold denotes significance at at least the α<0.05 level. * = p <0.05, † = p <0.01

Table 5.3-11 - ANCOVA of lactation duration in apes and non-apes

	Different slopes t_{111} *(p)*	Same slopes t_{111} *(p)*
Intercept	5.64 (<0.001***)	6.15 (<0.001***)
Body size	9.15 (<0.001***)	8.63 (<0.001***)
Ape	2.22 (<0.05*)	1.69 (0.09)
Body size * ape	-1.92 (0.06)	-
Lambda	.60	.64
R^2	.47	.44
AIC	-64.87	-63.18

ANCOVA model: response variable = lactation duration, predictors = body size (covariate) + ape. n=119, apes n=10.

Table 5.3-12 - ANCOVA of gestation duration in apes and non-apes

	Different slopes t_{134} *(p)*	Same slopes t_{134} *(p)*
Intercept	30.41 (<0.001***)	31.47 (<0.001***)
Body size	5.12 (<0.001***)	5.37 (<0.001***)
Ape	0.48 (0.63)	1.52 (0.13)
Body size * ape	-0.11 (0.92)	-
Lambda	.92	.92
R2	.19	.20
AIC	-429.33	-431.32

ANCOVA model: response variable = lactation duration, predictors = body size (covariate) + ape. n=146, apes n=13.

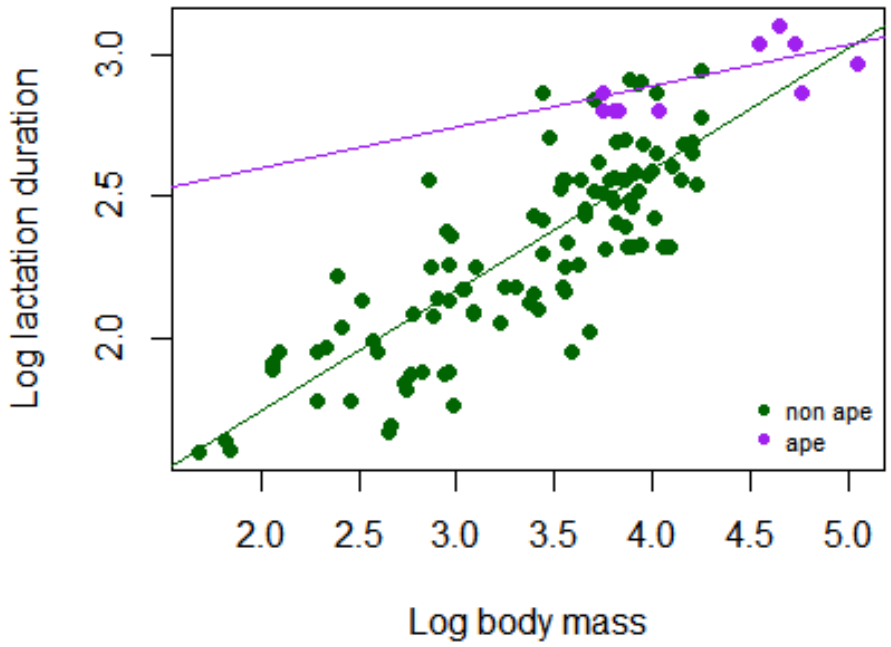


Figure 5:b - Different slopes ANCOVA of lactation duration in apes and non-apes

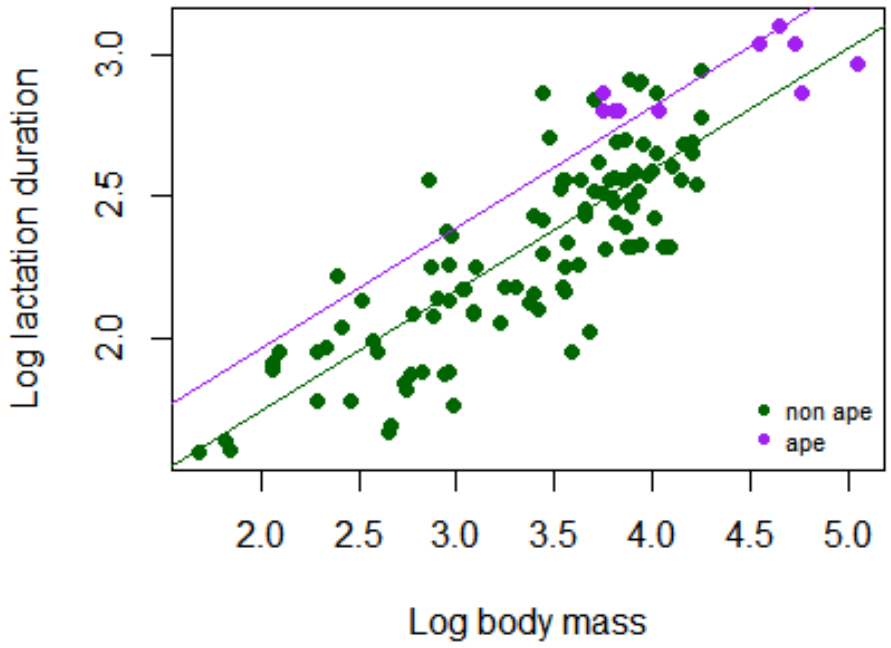


Figure 5:c - Same slopes ANCOVA of lactation duration in apes and non-apes

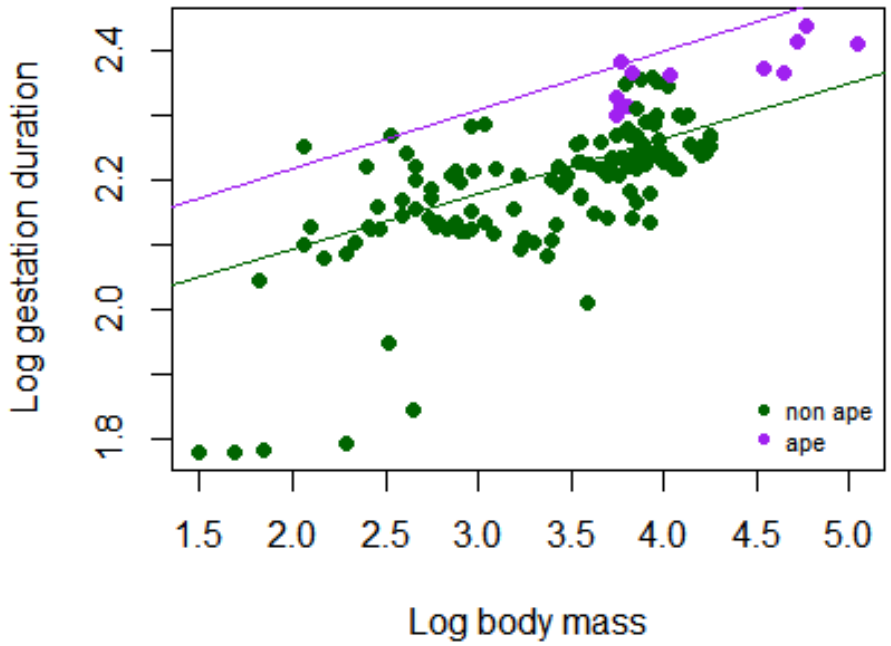


Figure 5:d - Different slopes ANCOVA of gestation duration in apes and non-apes

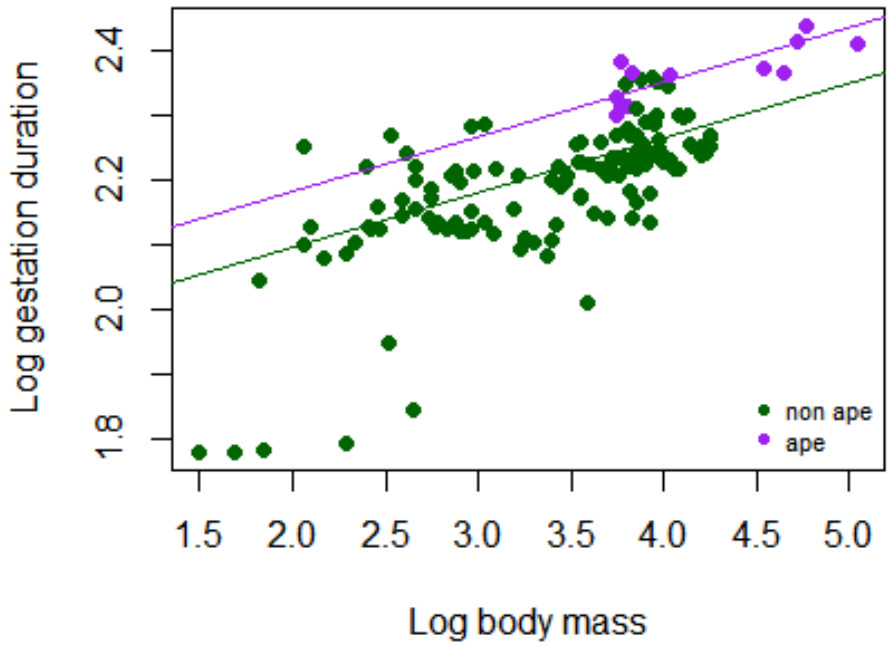


Figure 5:e - Same slopes ANCOVA of gestation duration in apes and non-apes

5.4 Discussion

5.4.1 Cognitive Buffer Hypothesis largely unsupported

Scant evidence was found to support a CBH interpretation of the evolution of brain or brain structure volumes. The central finding upon which the CBH was predicated, an independent correlation between brain size and longevity, was not found in this study. Rather, the apparent correlation between the two appears to be a by-product of a relationship between relative brain size and the duration of maternal investment, as previously found across Mammalia by Barton & Capellini (Barton and Capellini, 2011), but in contrast with more recent work on primates (Street *et al.*, 2017), which had a larger sample, but cruder neuroanatomical measurements (whole brain size) than the present study. Some proponents of the CBH have suggested that it is specifically the reproductive lifespan that should correlate with brain size, as this period is one of independence and the time during which behavioural flexibility, purportedly afforded by a large brain, should be relevant (Barrickman *et al.*, 2008). This study found no evidence to support this interpretation of the CBH either.

Rather than a general extension of lifespan in large brained species, this study found specific aspects of life history correlated with the volumes of different structures according to their developmental trajectories. Relative brain size appears to be governed primarily by developmental costs. Longevity is correlated independently of gestation duration, but this correlation is no longer present when lactation is controlled for. The absence of a specific correlation with either pre or postnatal investment and the log likelihood ratio test results showing that both gestation and lactation improve model fit could indicate there is insufficient statistical power to separate these effects, but it could also suggest that it is the totality of maternal energy provision which relates to whole brain size. This finding may be explained by the findings of Dubman et al. (Dubman, Collard and Mooers, 2012), who suggested that the overall duration of maternal investment is governed by metabolic rate, but the ratio of time within this period devoted to gestation or lactation is related to other "as yet unidentified, body size related" factors. An interaction between body size and lactation duration on cerebellum volume in the apes as suggested by the different slopes ANCOVA possibly supports their suggestion that this ratio is governed by constraints associated with body size, but the ANCOVA should be interpreted cautiously due to the lack of statistical power caused by the small number of apes.

Maternal investment, specifically prenatally, has an independent relationship with the relative volume of the whole brain and of 3 of the 4 structures investigated. This finding supports the contention that the size of neural structures is primarily governed by developmental factors, and that these constraints are related to the duration of gestation. However, there is variation in the life history correlates of structure volumes, which is explored below.

5.4.2 Neocortex

As predicted, only prenatal maternal investment showed a correlation with neocortical volume. Since the neocortex is composed of many heterogeneous systems, it is possible that other life history traits correlate with certain neocortical components, but these may not be well recovered when it is treated as a single structure. Indeed, developmental scheduling varies across the neocortex, with occipital grey matter maturing earlier than that in the prefrontal areas (Gilmore *et al.*, 2007). It is possible that those areas and tissues which continue to grow postnatally are associated with other life history variables like lactation and juvenile period. The disparity between the life history correlates of the cerebellum and neocortex is perhaps surprising given their demonstrated coevolution (Whiting and Barton, 2003; Herculano-Houzel, 2010; Smaers, Steele and Zilles, 2011; Barton, 2012; Lent *et al.*, 2012; Barton and Venditti, 2014).

5.4.3 Striatum

Striatum volume was not associated with postnatal maternal investment, contrary to the predictions based on the postnatal growth of some of its composite structures in humans. This could be due to different developmental scheduling in other parts of the striatum making a signal for the whole structure hard to detect, or it might suggest that the human pattern of striatal development on which the predictions were predicated are not generalisable across the primate order. Ernst and colleagues (Ernst *et al.*, 2014) found that humans generate striatal interneurons in to adulthood in a way which they suggest is unique to humans. This could explain the reported postnatal increase in caudate nucleus volume (Knickmeyer *et al.*, 2008), but the reported decrease in the relative (to brain size) volume of other striatal structures between 8 and 30 years (Ostby *et al.*, 2009) suggests that either this adult neurogenesis is not reflected by an increase in volume, or variability in the volume of these structures is different to that in other parts of the striatum, such as the caudate nucleus and olfactory tubercle. It is difficult to get a picture of overall striatum development as the literature frequently only

examines some of its constituent structures, so it is difficult to say whether observed developmental schedules are generalisable to the whole striatum.

The possible relationship between striatum volume and juvenile period is interesting given the late growth of the caudate nucleus referenced above (Knickmeyer *et al.*, 2008) and its role in movement; both traits that it shares with the cerebellum. Such a relationship may indicate that the growth of this structure is particularly sensitive to environmental exposure and social learning (see section 5.4.5.1 below). It is interesting to note the lack of an association with lactation coupled with the positive direction of the juvenility association, despite the reported increase in caudate volume in early postnatal life and the decrease in other striatal structure volumes later in life. On balance, the striatum results probably suggest that there is currently insufficient comparative literature on the volumetric pattern of development in the striatum as a whole to draw reliable predictions about its life history correlates, so a focus on this structure in future research may be valuable

5.4.4 Hippocampus

The hippocampus did not show any significant life history associations. Since there was evidence to show that the human hippocampus did not vary significantly in size postnatally (Knickmeyer *et al.*, 2008), it was reasoned that the growth of this structure was mostly completed prenatally, and so its volume was predicted to correlate with gestation duration. Previous work on the developmental and life history correlates of the hippocampus present a mixed picture. Amrein and colleagues found an association between hippocampal neurogenesis cessation and lifespan across a range of rodent and primate taxa, and thus across a range of life history patterns (Amrein, Isler and Lipp, 2011). In contrast, an early examination of the life history correlates of structure sizes found that hippocampus volume correlated with female age at first parturition but not longevity in primates (Allman, Mclaughlin and Hakeem, 1993), however these early analyses were non-phylogenetic and used residuals as data to correct for allometry, either of which can skew findings (Freckleton, 2002, 2009). In addition, there is some discord in the literature regarding the extent or even existence of adult hippocampal neurogenesis in humans; historically disregarded, then supported (Snyder, 2018), then very recently again discredited (Sorrells *et al.*, 2018). The overall picture of growth in the hippocampus is therefore difficult to characterise. The results of this study which found no hippocampal life history correlates may reflect the existence of different patterns of growth in different taxa according to as yet unclear neurodevelopmental factors.

5.4.5 Cerebellar associations

Cerebellum volume shows a different profile to the other structures. An extended juvenile period seems to be a significant correlate of the evolution of enlarged cerebellar size. Its relationship with postnatal maternal investment and juvenile period and lack of independent relationship with gestation period stands in contrast to all other structures examined and relative brain size. This fits with evidence indicating late volumetric maturation of the cerebellum, extending through infancy and beyond in humans (Knickmeyer *et al.*, 2008; Tiemeier *et al.*, 2010; Wu, Chen and Shen, 2011). As the cerebellum is quite small relative to whole brain size, it seems unlikely that the postnatal growth of the cerebellum is necessary to overcome obstetric constraints. Rather, it suggests an adaptive reason for the cerebellum to undergo postnatal maturation.

5.4.5.1 Juvenile period

The positive association between juvenile period and cerebellum volume was independent of the duration of adulthood (reproductive period). This, coupled with the lack of a correlation with longevity, supports specifically the juvenile learning payoff interpretation of the CBH in that it is specifically juvenility which demonstrates a relationship, rather than a general elongation of life or an elongation of adulthood during which unpredictability is buffered.

The cerebellar associations are therefore specifically postnatal but pre-maturational. This could suggest that exposure to the extrauterine environment is important for cerebellar development. The postnatal genesis of the majority of cerebellar granule cells indicates high functional plasticity during this time, making environmental stimuli all the more potent in the shaping of this structure (Kiessling *et al.*, 2014). The cerebellum is increasingly being shown to mediate a wide range of cognitive functions (Ramnani, 2006; Barton, 2012). The postnatal maturation of this structure may allow for environmental influence on the development of these kinds of faculties. Joffe suggested that primates' extended juvenile period has been selected for by pressures associated with (amongst other factors) social learning of foraging skills, and play-facilitated learning specifically (Joffe, 1997). Infancy and (probably to a greater extent) juvenility are periods of social learning, practice and play in an environment of reduced risk (Burghardt, 2010). Social play has been shown to correlate with cerebellum (Lewis and Barton, 2001) and neocortex ratio (Lewis, 2000). Kerney et al. reported a positive association between the relative volume of the largest components of the corticocerebellar

system and the proportion of a spcies' time budget spent in play, and also found significant differences in specific component volumes between the most and least playful species (Kerney *et al.*, 2017). Play has also been shown to correlate positively with both postnatal brain growth and behavioural flexibility (Montgomery, 2014). This represents an in vivo test of the assumptions of the juvenile learning payoff CBH: skills gained pre-adulthood translate in to benefits in adulthood. The specific association of cerebellum volume with juvenile period therefore points towards the potential importance of this early social exposure. A number of studies have emphasised the role of postnatal environmental and social stimuli in shaping neural connectivity (Sakai *et al.*, 2011; Miller *et al.*, 2012).

5.4.5.2 Cerebellum and adult lifespan

The absence of an association between cerebellar volume and adult lifespan suggests that behavioural flexibility gained from extended learning opportunities during juvenility do not necessarily translate directly into a longer lifespan, contrary to the central thesis of the juvenile learning payoff CBH. While it could be argued that we wouldn't necessarily expect an effect of adulthood to be independent of juvenile period if the extension of the latter begets the extension of the former, the additional absence of a correlation between cerebellum volume and post weaning lifespan suggests the two phases are somewhat independent. The question then follows; with no accompanying extension of either total lifespan or adulthood, what is the fitness benefit of a coevolved extended juvenile period and enlarged cerebellum? Perhaps the skills learned during an extended juvenile period might foster higher fertility through improved foraging or social skills, but a more parsimonious explanation might be that this pattern is just an extension of the developmental costs hypotheses. The fact that the association with juvenile period just misses significance after lactation duration is controlled for ($p=0.06$) suggests that developmental costs are still important. It is possible that any benefits in terms of increased opportunity to learn or deploy skills afforded by an enlarged cerebellum are still subject to constraints by fundamental developmental ceiling effects associated with brain and body size (Finlay and Darlington, 1995). Maternal costs may also not be confined to pre-weaning. Juvenility represents a time when primates are not fully independent; relying on provisioning and protection from others (Kaplan *et al.*, 2000; Leigh, 2004). The provisioning of a subadult who is not yet an efficient forager is a further maternal cost that seems to be largely overlooked in the literature examining life history correlates of brain size

5.4.5.3 Differences between apes and non-apes

When apes were removed from the PGLS analyses, the effects of lactation and juvenile period duration on cerebellum volume were absent. The results of the phylogenetic ANCOVA on lactation duration tentatively suggest apes have a different lactation profile to the non-apes. There is also potential evidence for an interaction between clade and body size. This is difficult to interpret as the apes in the sample are few in number, have a restricted range of body sizes (i.e. they are all relatively large), and the interaction just misses significance (p=0.058). It is not simply the case that apes have longer lactation durations for *any* body size, but within the range of the data where apes are sampled, they appear to have longer lactation durations relative to body size than non-apes (Figure 5:b). The model has limited explanatory power and these results must be treated as a preliminary treatment of the question given the available data. Taken together, these results may suggest that the relationship between cerebellar size and postnatal maternal investment is unique to the ape clade. The absence of any suggestion of difference in relative gestation duration between the ape and non-ape clades contrasts with the lactation duration results and somewhat strengthens support for a different lactation strategy in the apes which is independent of other aspects of life history. Apes have unusually large cerebella (Barton and Venditti, 2014) and particularly extended periods of immaturity (Kelley, 2004) with delayed locomotor independence (Young and Shapiro, 2018). This clade also shows high levels of social learning (van Schaik and Burkart, 2011) and play (Ramsey and McGrew, 2005). These findings may also suggest that rather than being born with relatively larger cerebella, the duration of postnatal growth of this particular structure is extended in Hominoidea.

5.4.6 Life history and ecology may be confounded

Some life history correlates may not be immediately evident in this analysis because they are masked by intermediary variables. As explored earlier in the thesis, social and behavioural-ecological variables show complex, interdependent correlations with brain structure sizes. Behavioural ecology could potentially drive associations between life history and brain size by influencing both. Effects of cognitive buffering are difficult to separate from the effects of behavioural-ecological variables as both often largely affect an animal during independence (i.e. post sexual maturity). One such variable is diet. Although diet undoubtedly has effects on brain size and composition across the life history, its role as a selective pressure on brain size arguably becomes significant post-weaning when some degree of independence in feeding is reached. Prior to this, the effect of diet is primarily as a constraint, as during

lactation and gestation there is a provision of energy which is free of cognitive demands (sourcing, processing, competition etc.). A number of researchers have assessed this issue (Ross and Jones, 1999; Deaner, Barton and van Schaik, 2002) and found no evidence for relationships between socioecological variables and life history traits which could confound life history – brain relationships. However, these studies are not recent and use techniques which have since been superseded such as independent contrasts and residuals as data (refer to Chapter 1 for discussion of issues surrounding this)

5.4.7 Limitations of the study

A limitation of this study in terms of examining the relationship between life history and neurodevelopment is the use of adult structure volumes, as rate of growth rather than size at maturity may be the important factor. Barton and Capellini (Barton and Capellini, 2011) found that precocial mammal species have a higher rate of brain growth than their altricial counterparts, indicating that the duration of growth is not the only variable which can contribute to brain structure size. Unfortunately, available comparative neurodevelopmental data at the level of individual structures is currently very limited.

Volumetric measures can provide only a very broad picture of growth in the brain. A major question that bedevils all volumetric brain analysis is: what does an increase in volume actually mean? Changes in rate of neurogenesis or myelination, the size of neurons, and the ratios of white to grey matter can all cause variation in volume. Volumetric expansion and shrinkage can also occur heterogeneously across different areas of a structure across its development, as seen in the neocortex (Brown *et al.*, 2012) . In the context of the present study, extended growth duration of a structure in one taxon does not necessarily translate to a larger volume than a taxon with shorter growth duration. More detailed histological investigations across the range of development in a number of species will allow for more specific hypotheses to be formed about the timing and nature of mosaic growth in the primate brain (Huttenlocher and Dabholkar, 1997).

5.4.8 Concluding remarks

Overall, developmental costs seem to best explain the pattern of correlations between primate brain structures and life history. Most structures followed a common pattern of development shared with relative brain size, but there is also evidence of variation in their life history correlates. Most notably, the cerebellum is linked specifically to infanthood and juvenility, perhaps signalling that exposure to the environment is important for growth in this structure.

Overall, the variation in the life history correlates of structure sizes suggests than selection on particular functional capacities causes developmental shifts which facilitates neural changes.

6 Summary and conclusions

This thesis sought to re-examine the big ideas in brain evolution, which have been dominated comparative analyses of whole brain volume, in the light of newer ideas about mosaic brain evolution using updated techniques and better, more recent data. To this end, I used phylogenetic comparative techniques to examine the evolution of the primate brain and the behavioural, ecological, social, and developmental correlates of brain size and composition. The aims and major findings of each chapter are summarised below, followed by discussion of the limitations of this work, reflections for further work, and final concluding remarks.

6.1 Summary of empirical chapters

6.1.1 Chapter 2 – the multivariate structure of the mammalian brain

Chapter 2 was an exploratory investigation of how individual brain structures vary in the mammals. Phylogenetic principal components analysis and phylogenetic least squares regression were employed to examine the similarities and differences in brain composition between primates, bats, and "insectivores" (now recognised to be a polyphyletic taxon). This chapter explored possible patterns with reference to the concerted (Finlay and Darlington, 1995) versus mosaic (Barton and Harvey, 2000) evolution debate. The pPCA results could not distinguish between the two models. In the pPCA analyses of both mammals and primates, a first component representing size explained the vast majority of the variance (~93%), supporting a concerted evolution interpretation. However, there were different patterns of variation in structure volumes between the three orders, contradicting Finlay and Darlington's claim that all mammals lie along the same allometries (Finlay and Darlington, 1995). The grouping of the primates in the morphospace was quite distinct from the other two orders, occupying a position which represented large body size and small main olfactory bulb. PGLS analyses of neocortex and cerebellum volume further demonstrated the differences between the orders, with different partial correlations in each order. The previously reported correlated evolution between the neocortex and cerebellum in mammals (Barton, 2012) was recovered, however when each order was analysed separately, the association was not evident in the "insectivores". Overall, these differences among clades did

not support a universal mammalian pattern of brain structure volume variation according to body size, but size related variation was certainly a significant force in shaping brain composition. Therefore, this chapter showed broad agreement with the literature in supporting a mosaic pattern of brain evolution with developmental aspects playing a smaller role in ultimate brain composition.

6.1.2 Chapter 3 – Re-evaluating the link between brain size and behavioural ecology in primates

Chapter 3 sought to re-examine the major hypotheses concerning the behavioural-ecological correlates of brain size with new data and improved methods. To examine the role of data quality and causes of disagreement in the literature, two large behavioural-ecological datasets were analysed in the same way whilst maintaining the same response variable, phylogeny and methods. This formally tested a proposed (Borries *et al.*, 2016) major cause of equivocality in comparative studies; instability resulting from variable data quality. PGLS and subsequent model comparisons showed a divergence in results between the two datasets, even when the species sample was identical. Analysis of the Stephan (1981) dataset which has been so widely utilised in comparative brain evolution enquiry suggested that the correlation between group size and brain size which formed the basis of Dunbar's SBH was dependent on using this sample. Little support was found for the Social Brain Hypothesis as defined by Dunbar (Dunbar, 1998; Dunbar and Shultz, 2007a). Overall, more support was found for ecological correlates of brain size than social, particularly in the case of a positive association with home range size. However, it was difficult to isolate their independent effects, possibly due to their combined influence in the form of "adaptive syndromes" (Nunn and van Schaik, 2002).

6.1.3 Chapter 4 – The behavioural ecology of structure sizes

Whole brain size has been criticised as a measure of limited utility due to the functional heterogeneity of the brain (Healy and Harvey, 1990; Logan *et al.*, 2017). Chapter 4 therefore attempted to examine the behavioural-ecological correlates of brain variation at a finer scale by focusing on specific brain structures, testing whether and how they are more functionally specific. A new dataset which augments and updates the Stephan (1981) dataset was used, providing data collected with more modern methods and a more representative sample. Six brain structures were chosen for analysis based on data availability to give the largest possible comparative sample. A composite variable of combined neocortex and cerebellum volume was also included to examine the correlates of this coevolved (Whiting and Barton,

2003) system. As in Chapter 3, stability was also examined across two behavioural-ecological datasets. Predictions for associations between structures and behavioural-ecological variables were made based on three prominent brain evolution hypotheses: the Ecological Brain (Clutton-Brock and Harvey, 1980; DeCasien *et al.*, 2017), the Social Brain (Dunbar, 1992; Dunbar, 1998; Dunbar and Shultz, 2007a) and the Visual Brain (Barton, 2007).

The instability uncovered in the previous chapter remained, with results varying across the datasets. Despite this, there was evidence of some stable effects. A remarkably stable and independent association between the thalamus and group size was an unpredicted outcome of this chapter. A pattern of swapping of significant effects between home range size and group size was discussed as possible evidence for their membership of an adaptive syndrome or possibly their correlation with a latent population density variable. Home range size results were again notable; this time showing associations with multiple structures. Results also varied, however, depending on correction for remaining brain size: one of the datasets showed associations between a number of structures and home range size when the size of the rest of the brain was not corrected for, which were absent when it was. This was discussed as possible evidence for a role of distributed functional systems associated with ranging.

6.1.4 Chapter 5 - Life history correlates of primate brain structure volumes

Chapter 5 examined the life history and ontogenetic correlates of volumetric structure change. Four structures were chosen for analysis based on whether clear predictions could be derived from the literature on their developmental scheduling. Predictions from energy costs based hypotheses (Martin, 1996; Isler and van Schaik, 2009) and cognitive buffering hypotheses (Barrickman *et al.*, 2008; Sol, 2009; González-Lagos, Sol and Reader, 2010) were tested. Little evidence was found in support of the cognitive buffer hypotheses. The correlation between longevity and brain size or brain structure size which forms the basis of cognitive buffer hypothesis (Allman, Mclaughlin and Hakeem, 1993; Allman, Mclaughlin and Hakeem, 1993) was not apparent once maternal investment duration was accounted for. Prenatal maternal investment was associated with the adult volume of 3 out of the 4 brain structures.

Previous work on the developmental scheduling of the human cerebellum had shown that it undergoes a period of intense volumetric growth postnatally, increasing by around 280% in absolute terms in the first year of life (Knickmeyer *et al.*, 2008). Based on this pattern of

strong postnatal growth, the cerebellum was predicted to correlate with postnatal maternal investment duration independently of overall maternal investment duration and lifespan. This prediction was supported; lactation was significantly associated with adult cerebellum volume, independently of gestation duration, demonstrating that cerebellum size is linked primarily to postnatal investment, rather than overall maternal investment. An association with the duration of the juvenile period (defined as the period between weaning and first parturition) was also recovered. The association was independent of the duration of the reproductive lifespan. This was supportive of an interpretation of the cognitive buffering hypothesis which emphasised the importance of the extension of the juvenile period for learning (Barrickman *et al.*, 2008), rather than the extension of the adulthood for either deployment of the learned skills or increased reproductive opportunities.

Since apes have been shown to have significantly larger cerebella than expected from their neocortex size relative to other anthropoid primates (Barton and Venditti, 2014), and also extended postnatal maturation (Leigh, 2004; Isler and van Schaik, 2012) their possible position as outliers and their consequent influence on these results was evaluated. Their removal from the PGLS investigations led to the absence of the lactation and juvenile period associations. An ANCOVA suggested that apes may exhibit a different lactation strategy from the rest of the primate order in line with their cerebellar expansion, but these results should be interpreted with caution due to the small number of ape species and resulting lack of statistical power.

6.2 Limitations

6.2.1 Volumetric data and distributed systems

This thesis has relied upon volumetric measures of brain and brain structure "size" due to the relative scarcity of comparative data on more fine-grained neuroanatomical measures such as neuron number. As previously discussed in the thesis introduction, the "size" of the brain has long been tacitly accepted as a proxy for cognitive or computational capacity (Healy and Rowe, 2007). However, what "size" means in terms of computation is not straightforward. The most fundamental unit of computation is generally thought to be the neuron (Herculano-Houzel, 2010; Mota and Herculano-Houzel, 2014), and larger brains or structures have been assumed to have greater numbers of neurons, and so greater computational capacity. This is

accurate for primates in absolute terms (Herculano-Houzel *et al.*, 2007), however brain and brain structure size may not scale with neuron number in the same way across clades (Herculano-Houzel, 2009, 2011). Scaling of neuron number with size also varies across structures within the same species (Herculano-Houzel, Manger and Kaas, 2014) and across areas of the same structure (Herculano-Houzel *et al.*, 2008; Ribeiro *et al.*, 2013). Neuron size also varies (Herculano-Houzel, Manger and Kaas, 2014; Mota and Herculano-Houzel, 2014), so that fewer, larger neurons may occupy more space than a greater number of smaller cells, reducing neuron density but possibly increasing the volume of the structure. Variation in the volume of a structure is therefore difficult to interpret. A unit change in the volume of a structure in two different clades does not necessarily indicate a common change in cytoarchitecture, capacity or computation.

In addition to uncertainty regarding the meaning of volumetric size, the way in which we commonly divide the brain may also be obstructing our efforts to understand its variation. Anatomically defined structures like those investigated in this thesis are potentially of limited utility in investigating the selective pressures and constraints which have contributed to their relative size. This is because the systems which mediate the functions upon which selection acts are distributed across these structures (Smaers, Steele and Zilles, 2011; Buckner and Krienen, 2013; Mars *et al.*, 2013). Therefore, even at the level of the structure, functional specificity is still lacking. Variation in these functionally distinct systems is therefore likely to be more robustly linked to specific behavioural, ecological and social correlates (Montgomery, Mundy and Barton, 2016). This was anticipated and somewhat parried by examining the covariation of structures and correlates of a compound cortico-cerebellar variable.

Incorporating distributed systems and more valid measures of computational or cognitive capacity like neuron density and size in to future work will doubtless yield more stable and reliable results than volumetric analyses. However, to achieve the sort of large scale comparative analyses necessary to give sufficient statistical power to give meaningful results requires large samples. Collection of such data would represent a huge research effort. Whilst comparative data on neuron densities and functional systems is emerging, it is still limited in its taxonomic scope (for example, neuron number estimates using consistent methods are available for around 40 mammal species (*http://www.suzanaherculanohouzel.com/*, 2018)).

6.2.2 Correlated predictors and syndromes of traits

Predicted relationships between behavioural-ecological variables and brain or brain structure size were largely not supported in Chapters 3 and 4. While this may be because the predictions are false, we must also consider possible evidence to suggest that the predictor variables used were not sufficiently representative of the concepts tested or that they were not sufficiently distinct from each other. A major issue for comparative studies of brain evolution is that predictors of brain size are frequently correlated (Barton, 1996; Walker *et al.*, 2006; Barrickman *et al.*, 2008; Schillaci, 2008; Swanson *et al.*, 2012; Weisbecker *et al.*, 2015), rendering their individual explanatory power difficult to ascertain (Healy and Rowe, 2007) and potentially leading to spurious correlations (Walker et al., 2006). An example of the latter was demonstrated by Nunn and Barton (Nunn and Barton, 2001) who found that the apparent relationship between group size and body size was mediated by the relationship of each with activity period and substrate use (p .87). This example also highlights the problem of disentangling an individual predictor's influence on the dependent variable from the other predictors it correlates with.

Taking a relevant example from this thesis; despite an apparent wealth of evidence in support of a relationship between brain size or neocortex size and social group size across mammal species (Dunbar, 1992; Barton and Dunbar, 1997; Pérez-Barbería, Shultz and Dunbar, 2007; Dunbar and Shultz, 2007a; Shultz and Dunbar, 2007), scant evidence of either was found in the analyses carried out in this thesis. Chapters 3 and 4 showed some evidence of a link between home range size and group size, which suggests their influence on brain evolution may also be linked. The two variables have been suggested to covary in the form of population density (Walker *et al.*, 2006). The number of animals per unit of space has obvious implications for food availability, competition for mates, predation, and terrestriality versus arboreality. It may therefore be variation in this joint variable, rather than the two individual variables, which has a stable influence.

Some of these variables may form syndromes of traits (Nunn and van Schaik, 2002); groups of interdependent, covarying variables which may have a combined influence on brain size and composition. This idea was proposed by Nunn and van Schaik (2002), who, in their work to reconstruct the lifestyles of extinct primate taxa, observed that certain trait values often co-occur in a species, whilst some combinations of traits never coincide. They give an illustrative example (p. 170); species which specialise in eating tree gum are always arboreal, tend to be small and live in wooded habitats. Large bodied, terrestrial gummivores living in

open environments do not occur. This example demonstrates how traits co-occur and co-vary, but only within certain parameters, which means that they remain useful for examining general patterns.

A possibly more valid way of examining the behavioural-ecological traits of a species, then, is to cease to treat them as independent of each other and instead interpret them as syndromes. The challenge is how these syndromes can be meaningfully defined and measured, as it is difficult to objectively characterise the biological meaning of such ecological dimensions. Arguably the most appropriate way to examine the possible presence and influence of an adaptive syndrome is to use variable reduction techniques like PCA and Factor Analysis. In addition to the objectivity issue raised above, another difficulty is that every species occupies a niche that is unique in some aspect. Even sympatric species occupy different parts of the same niche. Therefore, a factor analysis may reveal a highly dispersed distribution of species across the morphospace rather than any pattern of grouping, which does not enable us to derive common rules or make predictions based on their relative positions.

6.2.3 Multiple testing

When a hypothesis is tested multiple times, the chance of a Type I error (false positive) can increase (Field, Miles and Field, 2012). This is because the probability of obtaining a result by chance accrues cumulatively with each test, as each independent test introduces a new possibility to find a positive result by chance. In chapters 4 and 5, the same response variables were subjected to several PGLS analyses with various combinations of predictors and using different predictor data sets. Ecological and life history variables were regressed against brain structure volumes, and each test was repeated for a different dataset. It was therefore necessary to consider whether correction for multiple testing was appropriate in these cases.

The logic of correction for multiple testing is based on the increased risk of making a Type 1 error where each test introduces a new, independent chance to obtain significance by chance. However, the PGLS tests carried out in chapters 4 and 5 are not independent of one another, and so the Type 1 error does not increase in this way. The response variables are not independent of each other as they are brain structure volumes which are correlated due to being part of (a) common system(s) which is(are) anatomically, functionally and developmentally linked. Similarly, the predictor variables are also non-independent because

they are the exact same ecological/life history variables across each model. The risk of an inflated Type 1 error rate is therefore reduced, as the less independent the tests are of each other, the less the error rate will increase. Further, the analyses in chapters 4 and 5 tested for stability across datasets and examined incidences where significant results were stable. Therefore, this replication reduces the probability of finding significant results. Based on the above, it was not clear whether any correction for multiple testing should be made, and if one were to be made, how it should be applied. As there is no unequivocal solution to this particular issue, no correction was applied.

6.2.4 Data quality and replicability

The most pressing and fundamental issue facing the kind of comparative work undertaken in this thesis is arguably that of data quality and replicability. As uncovered by the analyses in Chapter 3 and 4, results tend to be unstable in the face of minor changes to the data. To adequately test hypotheses whilst controlling for confounding factors often requires large sample sizes. The difficulty of composing large datasets of brain measures and behavioural, ecological, physiological and social variables for multiple species has meant heavy reliance on just a few studies (Healy and Rowe, 2007). The creation of new larger datasets very commonly means the aggregation of many smaller datasets from many different sources, all with individual biases (Smith and Jungers, 1997). This overlap in sources means replicating analyses using such new datasets risks replicating inherent biases.

The composition of the sample in terms of taxonomic range is the first potential source of variability. To take an example from the literature; home range size is reportedly a more significant pressure on brain size in Old World monkeys than in New World monkeys (Walker *et al.*, 2006). Analysis of a dataset with a disproportionately large number of Old World monkeys could therefore have a greater chance of recovering a relationship between brain size and home range size than one using a more evenly distributed dataset. Parker (Parker, 2015) points out taxonomic biases in Stephan's 1981 dataset (Stephan, Frahm and Baron, 1981), which includes only one (juvenile) gorilla specimen and no orangutans or bonobos (p.2). Chapter 3 supported Parker's criticism, finding that the sample was insufficiently representative to produce reliable results. Although Chapter 4 and 5 use a volumetric dataset which has been augmented with more up to date and reliable measures (Navarrete, pers. comm.), it is still heavily reliant on the earlier Stephan work. This was unfortunately unavoidable due to the lack of available data for taxonomic breadth of sample required.

A second challenge is that, as is common in comparative ecological work, this thesis does not account for intra-specific variation, collapsing this variation to a mean. The validity of these means would be questionable if the within-species variance for a particular trait was large relative to that between species. The example of activity period in *Lemur catta* is illustrative of the problem. *Lemur catta* has been uncontroversially recorded as diurnal in many datasets, including the two used in Chapters 3 and 4. However, evidence is emerging that suggests this categorisation may be due to an overreliance on captive samples or studies that do not sample across seasons (Parga, 2011; Donati *et al.*, 2013). This example shows the potential for variability within a species in a single variable and the consequent variable validity of a categorical allocation of a species' activity period.

This simplification can also cause statistical problems; the practice of reducing complex traits to single numbers makes finding spurious correlations much more likely (Deacon, 1990). The issue is not confined to the socioecological variables; brain size is treated as a fixed value, but it does vary across individuals and also within them under the influence of developmental and experiential factors (Healy and Rowe, 2007). Phylogenetic methods which allow for within species variation by incorporating the uncertainty in to the error term are available, but this inflation of the error term can cause problems with underestimating patterns of correlation in data (phylogenetic and between traits) (Ives, Midford and Garland, 2007). It also still ultimately relies on species means which, as discussed above, are potentially lacking in biological meaning. New methods which can explicitly incorporate multiple values for an individual species are in preparation, but data availability is still lagging behind.

Given these difficulties, comparative work stands to gain much from better data and methods. In the short term, the verification of results by testing multiple datasets, as in chapters 3 and 4, can flag replicability issues and bolster confidence in the robustness of results. In the longer term, comparative work must become much more collaborative in spirit, by improving data provenance information (metadata) and moving towards standardisation of methods of data collection and definitions of variables (Borries *et al.*, 2016). Finally, incorporating intra-specific variation in to comparative datasets will enable researchers to account for this variation when the tools to do so become reliable and accepted.

6.3 Future research

6.3.1 Hypotheses

It is possible to draw a number of specific hypotheses from the findings of this thesis for future research:

1. *The thalamus has an independent association with group size*

   The surprising finding in Chapter 4 that both absolute and relative thalamus size shows a robust positive association with group size warrants further investigation. Firstly, the independence of this relationship from other functionally linked areas should be tested. While the association found between thalamus volume and group size was independent of any such association with the neocortex, the neocortex is so large and functionally heterogeneous that such an association may be present but not evident when the neocortex is examined as a whole. It would be illuminating therefore to explore whether specific thalamo-cortical circuits show evidence of an association with sociality.

2. *Increased postnatal maternal investment is necessary for growing large cerebella*

   This hypothesis is supported by the findings of Chapter 5, but this may be a pattern which is unique to apes. Examining other taxa which have been shown to have large cerebella, such as odontocete cetaceans (Marino *et al.*, 2000; Montgomery *et al.*, 2013) and elephants (Maseko *et al.*, 2012; Herculano-Houzel *et al.*, 2014), would enable us to explore the possible way in which allometric constraints are overcome to achieve this mosaic expansion. Both of these taxa have not only large cerebella but also long juvenile periods and lactation durations (Lee, 1996; Oftedal, 1997) and so a link between the two as demonstrated in the apes seems possible. This would make an interesting addition to the concerted versus mosaic evolution debate, showing how developmental traits can be modified to allow for mosaic volumetric change in a specific structure.

3. *The relatively large ape cerebellum is a result of a prolonged period of postnatal cerebellar growth which is facilitated by an elongated postnatal maternal investment period*

   Although the analyses carried out in Chapter 5 gave results which were suggestive of the positive association between lactation duration and cerebellum volume, their lack

of statistical power due to a small sample precluded drawing firm conclusions. Obtaining data for a larger sample of apes (great and small) would give more reliable results and help to establish whether the apes have a significantly different lactation duration for their body size than the rest of the primates, and whether this is associated with the expansion of the cerebellum in this clade.

6.3.2 Wider areas for further investigation

In addition to the specific hypotheses above, the findings of this thesis also flag a number of wider issues which should be considered in future work.

6.3.2.1 Pressures associated with the expansion of the cortico-cerebellar system

While not the main focus of this thesis, the cortico-cerebellar system has been discussed and analysed in three of the empirical results chapters. This system was not found to be reliably associated with any of the explored behavioural-ecological or life history variables. To the contrary; the neocortex and cerebellum often correlated with different behavioural variables (Chapters 3 and 4), and the two had a divergent pattern of life history correlates (Chapter 5), suggesting they have different developmental scheduling as shown in the neurodevelopmental literature (Knickmeyer *et al.*, 2008; Wu, Chen and Shen, 2011) There are a number of possible explanations for this apparent lack of functional or developmental specificity, including the functional and anatomical heterogeneity of the system.

However, it is also possible that the pressures influencing the size and composition of the cortico-cerebellar system were not adequately captured in the variables examined in this thesis. The cerebellum has been linked with the temporal and spatial organisation of behaviour (Leggio *et al.*, 2001; Ramnani, 2006), and its volume has been shown to correlate with extractive foraging (Barton, 2012). Some species make use of food sources that have defensive adaptations; such as fruits encased in hard, indigestible shells, leaves with defensive spines, or insects that live in fortified nests. The extractive foraging hypothesis (Parker and Gibson, 1977; Parker, 2015) posits that the cognitive challenge posed by the processing of these protected food sources is linked to the elaboration of complex sensorimotor processes and enlarged brains.

The cerebellum's dense connectivity with the neocortex allows for the assimilation and processing of the variety of sensory and motor information required to carry out complex sequential behaviour (Leiner, Leiner and Dow, 1993; Ramnani, 2006). Sequential, syntactically organised, complex behaviours have been proposed as a major reason for brain

size increases and have been linked with the cerebellum (Reader and Laland, 2002; Leiner, 2010; Heldstab *et al.*, 2016). These behaviours are most often associated with food processing, either manually or through the use of tools (Parker and Gibson, 1977; Byrne, Corp and Byrne, 2001; Byrne, 2006, 2007; Sabbatini *et al.*, 2014). The relative expansion of the ape cerebellum in comparison to other primates and their more frequent use of tools and extractive foraging is suggestive of a link (Barton, 2012). A reasonable next step in determining the possible behavioural-ecological correlates of the corticocerebellar complex would therefore be to examine its relationship with extractive foraging and tool use. A future study may incorporate some measure of dexterity (Byrne, Corp and Byrne, 2001; Mangalam and Fragaszy, 2015; Benson-Amram *et al.*, 2016; Heldstab *et al.*, 2016) or forelimb use (Iwaniuk, Pellis and Whishaw, 2000; Sacrey, Alaverdashvili and Whishaw, 2009; Swanson *et al.*, 2012) (if appropriate to the taxon in question) to further understand the possible cognitive and neuroanatomical links between fine motor skills and the sequential organisation of behaviour. Examining the possible technical cognition correlates of variation in specific cerebellar nuclei and regions with different functionality and connectivity would also allow examination of the fine-scale, system-level changes associated with this kind of behaviour.

6.3.2.2 Causality

The analyses carried out in this thesis have been correlative, which has firm conclusions about the direction of causality. As discussed in Chapter 3, path analysis is becoming commonly used and results are often described using causal language (Lehmann, Korstjens and Dunbar, 2007; Dunbar and Shultz, 2007a; Fox, Muthukrishna and Shultz, 2017). However, since path analysis is also correlational, it too is precluded from assessing causality (Denis and Legerski, 2003). It is a tool for testing different hypotheses based on identifying direct and indirect relationships between predictor variables. One model may fit the data better than another, but we still cannot infer a causal relationship from a correlation. A possible solution lies in ancestral trait reconstruction (Schluter *et al.*, 1997; Pagel, 1999, 1999). This method can estimate trait values at ancestral nodes, enabling the user to estimate which traits preceded or succeeded which, thus giving a way of estimating causality (while acknowledging that temporal order is a necessary but insufficient condition required to conclusively demonstrate causality) (Fabre *et al.*, 2013). This approach could expand our understanding in terms of whether changes in brain size or composition preceded or succeeded changes in behavioural ecology or life history. For example, correlational data may tell us that invasion success is linked to a larger brain (Sol *et al.*, 2008), but it cannot tell

us whether an existing larger brain causes an animal to be more successful in terms of invasion, or whether invasion causes a subsequent increase in brain size, perhaps due to the need for behavioural flexibility in a new niche. One possible promising target for this method would be hypothesis 3 above; reconstructing the evolution of the possible elongation of lactation duration in the apes alongside their rapid cerebellar expansion (Barton and Venditti, 2014) may help to illuminate the possible developmental aspects of mosaic change in brain structures. For example, if the extension of postnatal maternal investment is necessary to provide sufficient energy to grow a large cerebellum, we might expect the modification of the former trait to have succeeded the latter temporally.

6.4 Concluding remarks

In broad summary, the investigations carried out in this thesis show that primate brain evolution has been characterised by mosaic changes associated with behavioural-ecological pressures against a background of size-linked developmental constraints. This mosaicism is also observable in the way in which developmental traits are associated with brain and brain structure size, as demonstrated in Chapter 5 by the distinct pattern of life history correlates in the cerebellum. Analysing the major hypotheses in brain evolution at the finer level of the structure has revealed that these hypotheses have specific implications for specific parts of the brain, and so whole brain analyses, while sometimes useful, only give us part of the picture. It is necessary to consider changes in individual brain structures and systems of structures to get full picture of how behaviour, ecology, sociality, and development truly interacts with the brain.

With regard to the correlates of neural variation; the highly correlated nature of behavioural-ecological variables makes their relative influences difficult to tease apart and perhaps suggests that treating them as independent predictors is not meaningful. It is clear that many social and ecological pressures have interacted with each other, exerting a combined pressure on structures which themselves have co-evolved as well as undergoing their own independent evolution. By statistically treating these variables as though they are independent of each other, we have perhaps not been asking the right questions in order to understand how evolution has shaped brains. In terms of correlates that describe a taxon's niche, syndromes of linked correlates with linked outcomes may be more appropriate. This non-independence of behavioural-ecological correlates of neural variation, the distributed nature of the systems

which mediate function and the complex influence of development suggests that attempting to carve up the factors shaping brain evolution in to singular pressures exerting their influence on singular dimensions of brain size is likely misguided.

As Barton (2012) points out;

> *"The idea that there likely to have been a wide variety of selection pressures on cognitive abilities, and a corresponding variety of neural evolutionary responses, has been rather lost in the current enthusiasm for monolithic explanations for the evolution of large brains,.."*

(p. 2097)

The picture that is emerging is more nuanced and much less polarised. Future comparative work on the factors influencing brain evolution should consider this more integrated, continuous structure of both selective pressures and the neural machinery they affect.

Appendices

Appendix 1 - PGLS regression examining the effects of five behavioural-ecological variables on endocranial volume with home range size excluded

	Data set 1 (n=144)		Data set 2 (n=104)	
Predictor	t_{138}	*p*	t_{98}	*p*
Intercept	-6	<0.001***	1.6	0.1
Body Size	20.2	<0.001***	7.2	<0.001***
Diurnality	2.5	<0.05*	-0.9	0.4
Terrestriality	0.4	0.7	1.3	0.2
Folivory	-2.3	<0.05*	0.3	0.8
Group Size	2.1	<0.05*	1.1	0.3
Model summary:				
ʎ	.986		1	
R^2	.8		.42	

Home range size was removed from the full models to see if it would have any effect on group size.
*p<0.05, **p<0.01, ***p<0.001

Appendix 2 - Model comparisons based on AIC and the log likelihood ratio test for dataset 1

Model	AIC	Δi	Log likelihood	?2	*p*
i) Null/Allometric (body size only)	-372.73	>2	190.37		
ii) Home Range Size	-381.58	>2			
iii) Group Size	-375.92	>2			
iv) Folivory	-375.85	>2			
v) Terrestriality	-370.8	>2			
vi) Activity Period	-377.73	>2	193.87	7	<0.01**
vii) Activity Period & Home Range Size	-386.43	**1.49**	199.22	10.7	<0.01**
viii) Activity Period, Home Range Size & Folivory	-386.92	**1**	200.46	2.49	0.11
ix) Activity Period, Home Range Size, Folivory & Group Size	-386.92	**(AICmin)**	201.96	3	0.08
x) Full	-386.06	**1.86**	202.03	0.14	0.71

All models included logged body mass as a covariate. Δi represents the difference between the minimum model AIC (AICmin) and the model (i). Figures in bold denote models with a Δi between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).
*p<0.05, **p<0.01, ***p<0.001

Appendix 3 - Model comparisons based on AIC and the log likelihood test for dataset 2

Model	AIC	Δi	Log	?2	*p*

			likelihood		
i) Null/Allometric (body size only)	-209.66	>2	108.83		
ii) Group Size	-209.01	>2			
iii) Activity Period	-211.43	>2			
iv) Folivory	-207.67	>2			
v) Terrestriality	-207.68	>2			
vi) Home Range Size	-216.35	**1.95**	113.17	8.68	<0.01**
vii) Home Range Size & Activity Period	-218.29	**(AICmin)**	115.15	3.95	<0.05*
viii) Home Range Size, Activity Period & Terrestriality	-216.38	**1.91**	115.19	0.09	0.77
ix) Home Range Size, Activity Period, Terrestriality & Folivory	-212.4	>2	115.20	0.01	0.91
x) Full	-212.4	>2	115.20	0.01	0.91

All models included logged body mass as a covariate. Δi represents the difference between the minimum model AIC (AICmin) and the model (i). Figures in bold denote models with a Δi between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).
*p<0.05, **p<0.01, ***p<0.001

Appendix 4 - PGLS regression examining the effects of five behavioural-ecological variables on endocranial volume with independent data sets

	Data set 1 (n=49)		Data set 2 (n=48)	
Predictor	t_{42}	p	t_{41}	p
Intercept	-5	<0.001***	10.4	<0.001***
Body Size	12.3	<0.001***	11	<0.001***
Diurnality	1.8	0.1	1.4	0.2
Terrestriality	0.9	0.4	0.7	0.5
Folivory	-2.5	<0.05*	-1.1	0.3
Group Size	1.9	0.1	-0.4	0.7
Home Range Size	1.5	0.1	1.1	0.3
Model summary:				
λ	.92		1	
R^2	.87		.82	

Each dataset was made completely independent by selecting species at random and including it in one and excluding it from the other. *p<0.05, **p<0.01, ***p<0.001

Appendix 5 - Model comparisons for the dataset 1 when species matched with dataset 2

Model	AIC	Δ_i
i) Full	-226.94	>2
ii) Null/Allometric (body size only)	-222.81	>2
iii) Home Range Size	-225.48	>2
iv) Group Size	-223.28	>2
v) Activity Period	-224.45	>2
vi) Folivory	-227.47	>2
vii) Terrestriality	-220.82	>2
viii) Folivory & Activity Period	**-228.93**	**0.8**
ix) Folivory, Activity Period and Home Range Size	**-229.69**	**(AIC_{min})**
x) Folivory, Activity Period, Home Range Size & Group Size	**-228.87**	**0.82**

All models included logged body mass as a covariate. Δi represents the difference between the minimum model AIC (AICmin) and the model (i). Figures in bold denote models with a Δi between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).

Appendix 6 - Model comparisons for dataset 2 when species matched with dataset 1

Model	AIC	Δ_i
I) Full	-196	>2
ii) Null/Allometric (body size only)	-195.81	>2
iii) Home Range Size	-200.16	**1.75**
iv) Group Size	-194.56	>2
v) Activity Period	-197.66	>2
vi) Folivory	-193.92	>2
vii) Terrestriality	-193.82	>2
viii) Home Range Size & Activity Period	**-201.92**	**(AIC_{min})**
ix) Home Range Size, Activity Period & Terrestriality	**-199.96**	**1.95**
x) Home Range Size, Group Size, Activity Period and Folivory	-196	>2

All models included logged body mass as a covariate. Δi represents the difference between the minimum model AIC (AICmin) and the model (i). Figures in bold denote models with a Δi between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).

Appendix 7 - Model comparisons for data set 1 after being made independent

Model	AIC	Δ_i
i) Full	**-98.88**	**1.1**
ii) Null/Allometric (body size only)	-84.4	>2
iii) Home Range Size	-92.39	>2
iv) Group Size	-94.7	>2
v) Activity Period	-86.37	>2
vi) Folivory	-88.75	>2
vii) Terrestriality	-82.71	>2
viii) Folivory & Group Size	**-99.14**	**0.85**
ix) Folivory, Group Size & Diurnality	**-99.75**	**0.23**
x) Folivory, Group Size, Diurnality & Home Range Size	**-99.98**	**(AIC_{min})**

All models included logged body mass as a covariate. Δ_i represents the difference between the minimum model AIC (AIC_{min}) and the model (i). Figures in bold denote models with a Δ_i between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).

Appendix 8 - Model comparisons for the data set 2 after being made independent

Model	AIC	Δ_i
I) Full	-85.01	>2
ii) Null/Allometric (body size only)	**-89.49**	**0.04**
iii) Home Range Size	**-88.91**	**0.63**
iv) Group Size	-87.54	>2
v) Activity Period	**-89.54**	**AIC_{min}**
vi) Folivory	**-88.97**	**0.57**
vii) Terrestriality	-87.95	>2
viii) Diurnality & Home Range Size	**-89.33**	**0.21**
ix) Diurnality, Home Range Size & Folivory	**-88.46**	**1.08**
x) Home Range Size, Group Size, Diurnality and Folivory	-86.85	>2

All models included logged body mass as a covariate. Δ_i represents the difference between the minimum model AIC (AIC_{min}) and the model (i). Figures in bold denote models with a Δ_i between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).

Appendix 9 - PGLS regression examining the effects of five behavioural-ecological variables on endocranial volume with species matched with the Stephan data set (Stephan, Frahm and Baron, 1981)

	Data set 1 (n=34)		Data set 2 (n=32)	
Predictor	t_{27}	*p*	t_{25}	*p*
Intercept	-5.7	<0.001***	9	<0.001***
Body Size	11.9	<0.001***	10	<0.001***
Diurnality	1	0.3	0.1	0.9
Terrestriality	-0.4	0.7	-0.6	0.5
Folivory	-1.2	0.3	-1.7	0.1
Group Size	1.8	0.1	1.8	0.1
Home Range Size	0.5	0.6	1.1	0.3
Model summary:				
ʎ	1		1	
R^2	.91		.89	

To determine whether previous results may have been affected by biases in Stephan's data set, the analysis was confined to only those species included in Stephan's original data set. *p<0.05, **p<0.01, ***p<0.001

Appendix 10 - Model comparisons based on AIC and the log likelihood ratio test for dataset 1 when species matched with the Stephan dataset (Stephan, Frahm and Baron, 1981)

Model	AIC	Δi	Log likelihood	⍰2	*p*
i) Null/Allometric (body size only)	-43.97	>2	23.984		
ii) Home Range Size	-45.4	>2			
iii) Activity Period	-45.5	>2			
iv) Folivory	-49.36	>2			
v) Terrestriality	-42.03	>2			
vi) Group Size	-51	>2	28.503	9.04	<0.01**
vii) Group Size & Folivory	-53.02	(AICmin)	30.512	4.02	<0.05*
viii) Group Size, Folivory and Home Range Size	-51.44	**1.59**	30.719	0.41	0.52
ix) Group Size, Folivory, Home Range Size & Terrestriality	-49.45	>2	30.727	0.02	0.9
x) Full	-48.91	>2	31.456	1.46	0.23

All models included logged body mass as a covariate. Δi represents the difference between the minimum model AIC (AICmin) and the model (i). Figures in bold denote models with a Δi between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).
*p<0.05, **p<0.01, ***p<0.001

Appendix 11 - Model comparisons based on AIC and the log likelihood ratio test for dataset 2 when species matched with the Stephan dataset (Stephan, Frahm and Baron, 1981)

Model	AIC	Δi	Log likelihood	?2	*p*
i) Null/Allometric (body size only)	-32.97	>2	18.489		
ii) Home Range Size	-36.78	>2			
iii) Activity Period	-32.29	>2			
iv) Folivory	-38.49	>2			
v) Terrestriality	-31.11	>2			
vi) Group Size	-39.7	>2	22.852	8.73	<0.01**
vii) Group Size & Folivory	-43.24	**AICmin**	25.622	5.54	<0.05*
viii) Group Size, Folivory and Home Range Size	-42.15	**1.1**	26.073	0.9	0.34
ix) Group Size, Folivory, Home Range Size & Terrestriality	-62.96	>2	26.361	0.58	0.45
x) Full	-38.73	>2	26.366	0.01	0.93

All models included logged body mass as a covariate. Δi represents the difference between the minimum model AIC (AICmin) and the model (i). Figures in bold denote models with a Δi between 0 and 2 and so are considered to have substantial empirical support (Kenneth P. Burnham and Anderson, 2002).
*p<0.05, **p<0.01, ***p<0.001

Appendix 12 - PGLS regression examining the effects of five behavioural-ecological variables on endocranial volume for data set 1 using sleeping group size where available

	(n=144)	
Predictor	t_{137}	*p*
Intercept	-5.4	<0.001***
Body Size	18.7	<0.001***
Diurnality	2.7	<0.01**
Terrestriality	0.6	0.6
Folivory	-1.6	0.1
Group Size	1.1	0.3
Home Range Size	2.6	<0.05*
Model summary:		
Λ	.99	
R^2	.8	

As the group size variable in data set 1 was a composite variable which was considered both foraging and sleeping group size, we ran the analysis again using only sleeping group size where it was recorded in the data set. *p<0.05, **p<0.01, ***p<0.001

Appendix 13 - PGLS regression examining the effects of five behavioural-ecological variables on endocranial volume for data set 1 using only species where body sizes were drawn from female animals

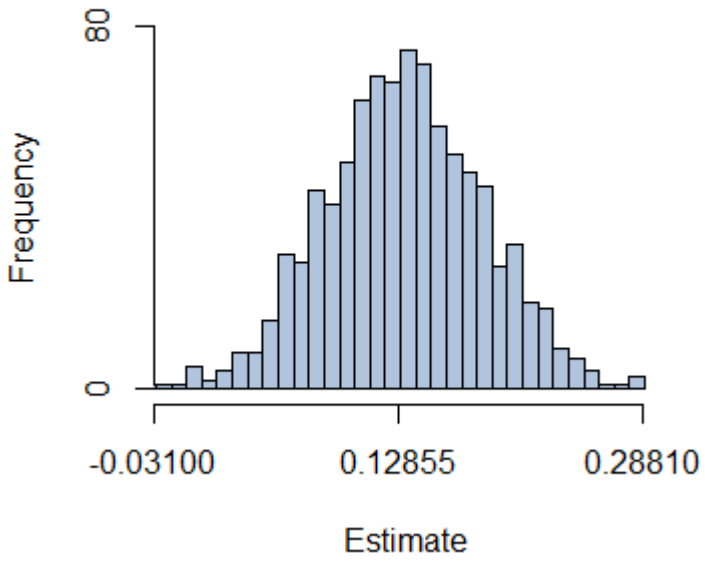
	(n=72)	
Predictor	**t_{66}**	***p***
Intercept	1.4	0.17
Body Size	10.8	<0.001***
Terrestriality	1.9	0.07
Folivory	-1	0.05
Group Size	0.9	0.4
Home Range Size	1.5	0.1
Model summary:		
λ	1	
R^2	.72	

As the body size data in data set 2 was drawn from exclusively female animals (in contrast to data set 1 which used means from males and females in some cases), we ran the PGLS analysis on the data set 1 again, using only the species with female body sizes. Activity period could not be included as all species were diurnal and so the variable had zero variance. *p<0.05, **p<0.01, ***p<0.001

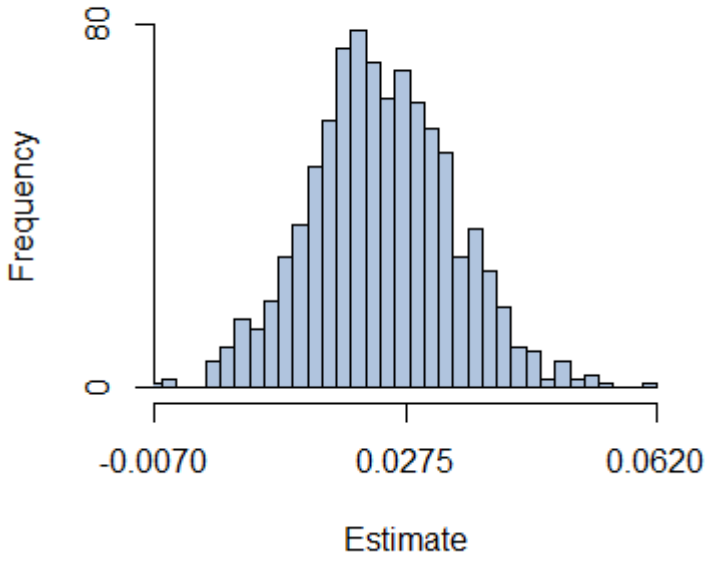
Appendix 14 - Data set 1 MCMC analysis diagnostics (n=145)

Predictor	Posterior mean	95% CI (lower)	95% CI (higher)	pMCMC
Body Size	0.5639	0.4991	0.6160	0
Diurnality	0.1327	0.0323	0.2174	0.0154
Terrestriality	0.008	-0.0267	0.0388	0.6725
Folivory	-0.0313	-0.0658	-0.0029	0.0637
Group Size	0.03567	-0.0133	0.0765	0.1408
Home Range Size	0.0247	0.0053	0.0406	0.0066

Appendix 15 - Posterior distribution of trait estimates for dataset 1- Activity period (diurnality)



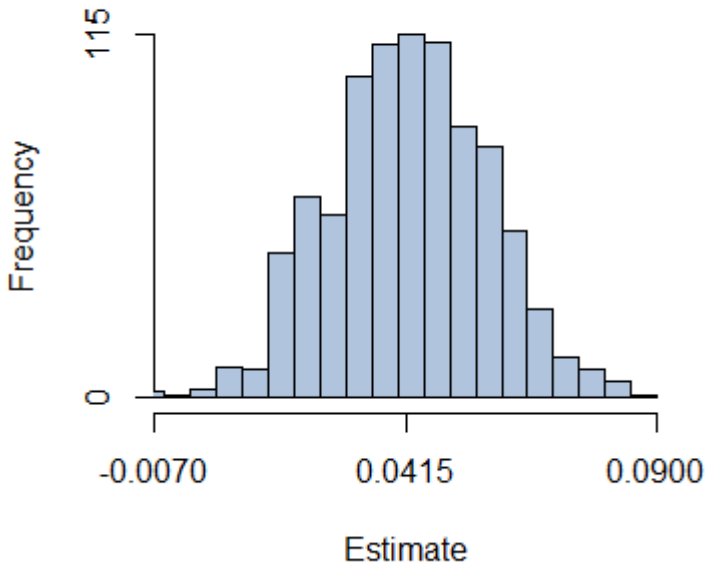
Appendix 16 - Posterior distribution of trait estimates for dataset 1- Home range size



Appendix 17 - Data set 2 MCMC analysis diagnostics (n=104)

Predictor	Posterior mean	95% CI (lower)	95% CI (higher)	pMCMC
Body Size	0.5396	0.4499	0.6076	0
Diurnality	0.1401	-0.0106	0.2705	0.0571
Terrestriality	-0.0075	-0.0572	0.0347	0.7538
Folivory	0.0017	-0.0386	0.0345	0.9319
Group Size	0.0017	-0.0674	0.0557	0.9341
Home Range Size	0.0426	0.0141	0.0674	0.0066

Appendix 18 - Posterior distribution of trait estimates for dataset 2- Home range size



Appendix 19 - PGLS regression on data set 1 examining the effects of five behavioural-ecological variables on endocranial volume when a more strict definition of folivory is used

	(n=144)	
Predictor	t_{138}	*p*
Intercept	-6.1	<0.001***
Body Size	20.4	<0.001***
Diurnality	2.6	<0.01**
Terrestriality	-0.1	0.9
Folivory	-3	<0.01**
Group Size	1.7	0.1
Home Range Size	2.6	<0.05*
Model summary:		
ʎ		.983
R^2		.82

Gorillas were not classified as folivores. Although *Gorilla* diet can include relatively large proportions of fruit, it is arguably a specialised folivore as its large size enable it to subsist on a leaf-rich diet when and where fruit is less available. We therefore ran two analyses, with Gorilla classified as non-folivore and folivore respectively. *p<0.05, **p<0.01, ***p<0.001

Appendix 20 - PGLS regression on data set 1 examining the effects of five behavioural-ecological variables on endocranial volume when a more strict definition of folivory is used

	(n=144)	
Predictor	t_{138}	*p*
Intercept	6.3	<0.001***
Body Size	20.5	<0.001***
Diurnality	2.6	<0.05*
Terrestriality	-0.1	1
Folivory	-3.2	<0.01**
Group Size	1.5	0.1
Home Range Size	2.6	<0.05*
Model summary:		
ʎ		.981
R^2		.82

Gorillas classified as folivores. *p<0.05, **p<0.01, ***p<0.001

Appendix 21 - paired t-test: Navarrete vs Stephan data

	t_{11}	p
Body size	-1.67	0.12
Brain volume	-1.8	0.1
Neocortex volume	-2.0	0.07
Hippocampus volume	-1.72	0.11
Striatum volume	-2.17	0.05
Cerebellum volume	-1.23	0.24
Medulla volume	-3.28	0.01**

*p<0.05, **p<0.01, ***p<0.001

Appendix 22 - Model comparisons for neocortex volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	R^2	p (R^2)[b]
Allometric (body size only)	-21.03	12.52		
Body size + activity period	-24.22	15.11	5.19	**<0.05***
Body size + diet	-25.20			
Body size + home range size	-27.18			
Body size + group size	-24.08			
Body size + activity period + diet	**-28.42**	18.21	6.2	**<0.05***
Body size + activity period + diet + home range size	**-29.92**	19.96	3.5	0.06
Body size + activity period + diet + home range size + group size	**-28.21**	20.11	0.29	0.59

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 23 - Model comparisons for hippocampus volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	R^2	p (R^2)[b]
Allometric (body size only)	**-33.25**	18.62		
Body size + activity period	**-34.47**	20.23	3.22	0.07
Body size + diet	-32.08			
Body size + group size	-32.82			
Body size + home range size	-31.31			
Body size + activity period + diet	**-34.15**	21.08	0.74	0.19
Body size + activity period + diet + group size	**-32.89**	21.45	0.74	0.39
Body size + activity period + diet + group size + home range	**-32.21**	22.11	1.32	0.25

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 24 - Model comparisons for striatum volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	R^2	p (R^2)[b]
Allometric (body size only)	-61.56	32.78		

Body size + group size	**-67.55**	36.78	7.99	**<0.01****
Body size + diet	-64.65			
Body size + home range size	-65.26			
Body size + activity period	-60.76			
Body size + group size + diet	**-67.38**	37.69	1.82	0.18
Body size + group size + diet + home range size	-64.41	37.21	0.96	0.33
Body size + group size + diet + home range size + activity period	-64.08	38.04	1.67	0.2

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
**p*<0.05, ***p*<0.01, ****p*<0.001

Appendix 25 - Model comparisons for cerebellum volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	□[2]	*p* (□[2])[b]
Allometric (body size only)	-78.21	41.1		
Body size + home range size	**-84.91**	45.46	8.71	**<0.01****
Body size + activity period	-77.34			
Body size + group size	-79.13			
Body size + diet	-77.57			
Body size + home range size+ activity period	**-83.57**	45.78	0.66	0.42
Body size + home range size + activity period + group size	-81.67	45.84	0.1	0.75
Body size + home range size+ activity period + group size + diet	-79.7	45.85	0.03	0.87

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
**p*<0.05, ***p*<0.01, ****p*<0.001

Appendix 26 - Model comparisons for cortex+cerebellum volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	□[2]	*p* (□[2])[b]
Allometric (body size only)	-51.96	27.98		
Body size + home range size	**-59.7**	32.85	9.73	**<0.01****
Body size + activity period	-54.41			
Body size + diet	-55.16			
Body size + group size	-56.56			
Body size + home range size + activity period	**-60.65**	34.33	3	0.09
Body size + home range size + activity period + diet	**-60.49**	35.25	1.84	0.18
Body size+ home range size + activity period + diet + group size	**-59.06**	35.53	0.57	0.45

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
**p*<0.05, ***p*<0.01, ****p*<0.001

Appendix 27 - Model comparisons for thalamus volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	□[2]	*p* (□[2])[b]
Allometric (body size only)	-60.59	43.4		
Body size + group size	**-62.96**	34.48	17.84	**<0.001*****
Body size + activity period	-58.67			
Body size + home range size	-60.42			

Body size + diet	-59.39			
Body size + group size + activity period	**-61.66**	34.83	0.7	0.4
Body size + group size + activity period + home range size	-59.87	34.93	0.21	0.65
Body size + group size + activity period + home range size + diet	-57.87	34.94	0.01	0.93

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 28 - Model comparisons for lateral geniculate nucleus volume in dataset 3 – body size corrected condition

	AIC[a]	Log likelihood	R^2	p (R^2)[b]
Allometric (body size only)	-52.05	28.02		
Body size + activity period	-56.18	31.09	6.13	**<0.05***
Body size + home range size	-60.32			
Body size + diet	-53.4			
Body size + group size	-55.96			
Body size + activity period + home range size	**-62.95**	35.47	8.77	**<0.01****
Body size + activity period + home range size + diet	**-62.5**	36.25	1.55	0.21
Body size + activity period + home range size + diet + group size	**-60.96**	36.58	0.47	0.5

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 29 - Model comparisons for neocortex volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	R^2	p (R^2)[b]
Allometric 1 (body size only)	-21.03	12.52		
Allometric 2 (body size + RoB)	-22.72	14.36	3.68	0.05
Body size + RoB + activity period	-25.6	16.8	4.88	**<0.05***
Body size + RoB + diet	-25.43			
Body size + RoB + home range size	-26.53			
Body size + RoB + group size	-25.05			
Body size + RoB + activity period + diet	**-28.61**	19.31	5.01	**<0.05***
Body size + RoB + activity period + diet + home range size	**-29.01**	20.5	2.4	0.12
Body size + RoB + activity period + diet + home range size + group size	**-27.39**	20.69	0.38	0.54

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 30 - Model comparisons for hippocampus volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	R^2	p (R^2)[b]
Allometric 1 (body size only)	-33.25	18.62		
Allometric 2 (body size + RoB)	**-39.19**	22.6	7.95	**<0.01****
Body size + RoB + activity period	**-38.4**	23.2	1.21	0.27
Body size + RoB + diet	**-39.47**			
Body size + RoB + group size	**-39.02**			
Body size + RoB + home range size	**-37.65**			

Body size + RoB + activity period + diet	**-39.3**	24.65	2.9	0.09
Body size + RoB + activity period + diet + group size	**-39.43**	25.71	2,13	0.14
Body size + RoB + activity period + diet + group size + home range	**-37.81**	25.91	0.38	0.54

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 31 - Model comparisons for striatum volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	χ^2	p (χ^2)[b]
Allometric (body size only)	-61.56	32.78		
Allometric 2 (body size + RoB)	-133.83	69.92	74.27	**<0.001*** **
Body size+ RoB + group size	-134.17	71.09	2.34	0.13
Body size + RoB + diet	-132			
Body size + RoB + home range size	-133.24			
Body size + RoB + activity period	-131.95			
Body size + RoB + group size + home range size	**-138.14**	74.07	5.97	**<0.05***
Body size + RoB + group size + home range size + activity period	**-136.45**	74.23	0.31	0.57
Body size + RoB + group size + home range size + activity period + diet	-134.5	74.25	0.04	0.83

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 32 - Model comparisons for cerebellum volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	χ^2	p (χ^2)[b]
Allometric (body size only)	-78.21	41.1		
Allometric 2 (body size + RoB)	**-168.52**	87.26	92.32	**<0.001*** **
Body size + RoB + diet	**-168.06**	8.03	1.54	0.21
Body size + RoB + activity period	**-167.2**			
Body size + RoB + group size	**-167.45**			
Body size + RoB + HRS	**-166.86**			
Body size + RoB + diet + group size	**-167.02**	88.51	0.95	0.31
Body size + RoB + diet + group size + activity period	-165.58	88.79	0.57	0.45
Body size + RoB + diet + group size + activity period + home range size	-163.59	88.8	0.01	0.93

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 33 - Model comparisons for cortex+cerebellum volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	χ^2	p (χ^2)[b]
Allometric 1 (body size only)	-51.96	27.98		
Allometric 2 (body size + RoB)	-54.19	30.1		
Body size + RoB + activity period	-56.3	32.15	4.11	**<0.05***
Body size + RoB + diet	-55.93			
Body size + RoB + home range size	**-59.28**			
Body size + RoB + group size	**-58.06**			
Body size + RoB + activity period + diet	**-58.01**	34.01	3.71	0.05
Body size + RoB + activity period + diet + home range size	**-59.74**	35.87	3.73	0.05
Body size + RoB + activity period + diet + home range size	**-58.43**	36.21	0.69	0.41

+ group size

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 34 - Model comparisons for thalamus volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	χ^2	p (χ^2)[b]
Allometric 1 (body size only)	-60.59	32.3		
Allometric 2 (body size + RoB)	-96.31	51.12	37.72	**<0.001*** **
Body size + RoB + group size	**-98.29**	53.15	3.98	**<0.05***
Body size + RoB + activity period	-94.37			
Body size + RoB + home range size	-94.38			
Body size + RoB + diet	-94.54			
Body size + RoB + group size + home range size	**-99.11**	54.55	2.81	0.09
Body size + RoB + group size + home range size + activity period	**-97.17**	54.59	0.06	0.8
Body size + RoB + + group size + home range size + activity period + diet	-95.18	54.59	0.01	0.94

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 35 - Model comparisons for lateral geniculate nucleus volume in dataset 3 – RoB corrected condition

	AIC[a]	Log likelihood	χ^2	p (χ^2)[b]
Allometric 1 (body size only)	-52.05	28.02		
Allometric 2 (body size + RoB)	**-77.03**	41.51	26.98	**<0.001*** **
Body size + RoB + activity period	**-77.47**	42.74	2.44	0.12
Body size + RoB + home range size	-75.49			
Body size + RoB + diet	**-77.21**			
Body size + RoB + group size	**-75.88**			
Body size + RoB + activity period + diet	**-77.71**	43.86	2.24	0.13
Body size + RoB + activity period + diet + group size	**-76.21**	44.1	0.49	0.48
Body size + RoB + activity period + diet + group size + home range size	-74.13	44.13	0.06	0.81

[a] Bold denotes best model and models with an AIC difference from the best model <2
[b] Bold denotes statistically significant model at the α<0.05 level
*p<0.05, **p<0.01, ***p<0.001

Appendix 36 - correlation matrix of predictor variables in dataset 1 (non-phylogenetic)

	Body size	Activity period	Group Size	Diet (folivory)	Home range size
Body size					
Activity period	**.49**				
Group Size	**.52**	**.56**			
Diet (folivory)	**.33**	.05	.06		
Home range size	**.7**	**.49**	**.7**	-.07	

Bold denotes statistically significant model at the α<0.05 level

Appendix 37 - correlation matrix of predictor variables in dataset 2 (non-phylogenetic)

	Body size	Activity period	Group Size	Diet (folivory)	Home range size
Body size					
Activity period	**.23**				
Group Size	.24	**.55**			
Diet (folivory)	**.31**	.08	-.05		
Home range size	**.5**	**.43**	**.73**	-.04	

Bold denotes statistically significant model at the α<0.05 level

Appendix 38 - correlation matrix of predictor variables in dataset 3 (non-phylogenetic)

	Body size	Activity period	Group Size	**Diet (folivory)**	**Home range size**
Body size					
Activity period	**.51**				
Group Size	**.51**	**.57**			
Diet (folivory)	**.22**	-.07	-.13		
Home range size	**.75**	**.55**	**.7**	**-.17**	

Bold denotes statistically significant model at the α<0.05 level

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